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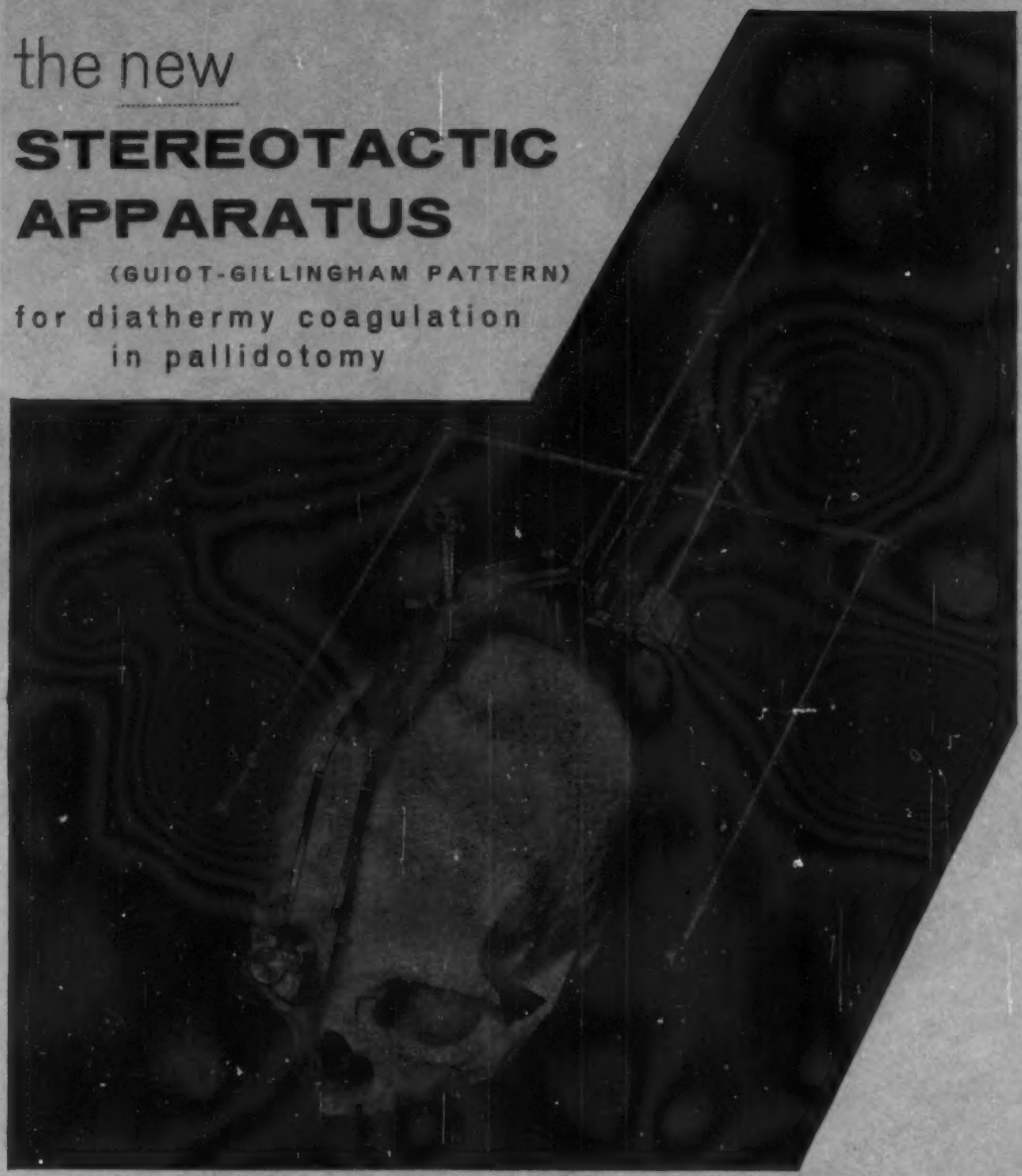

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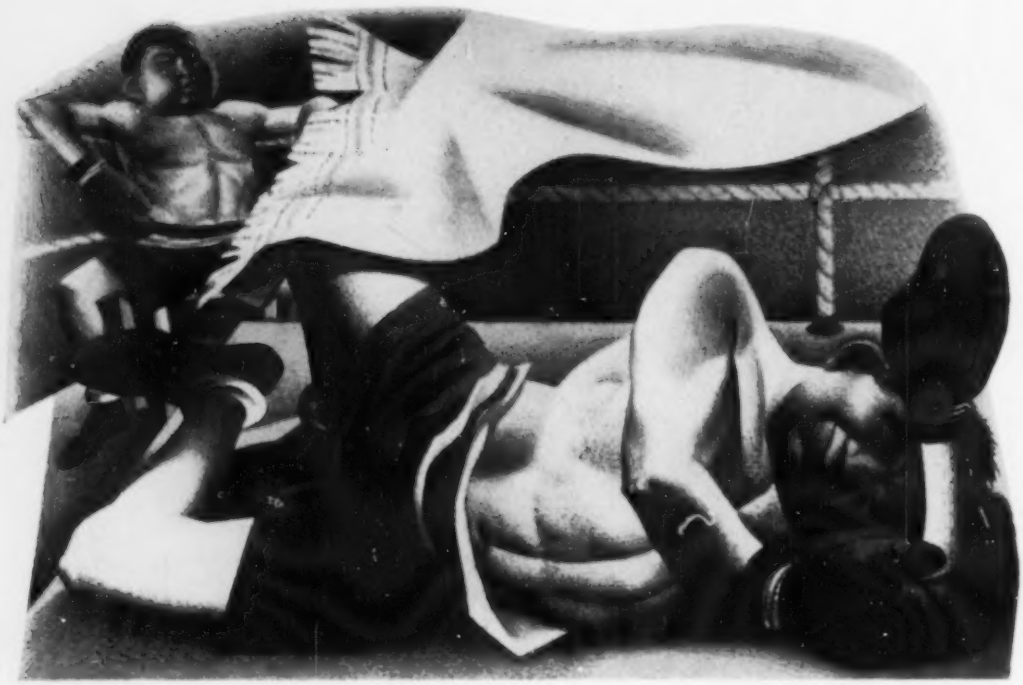

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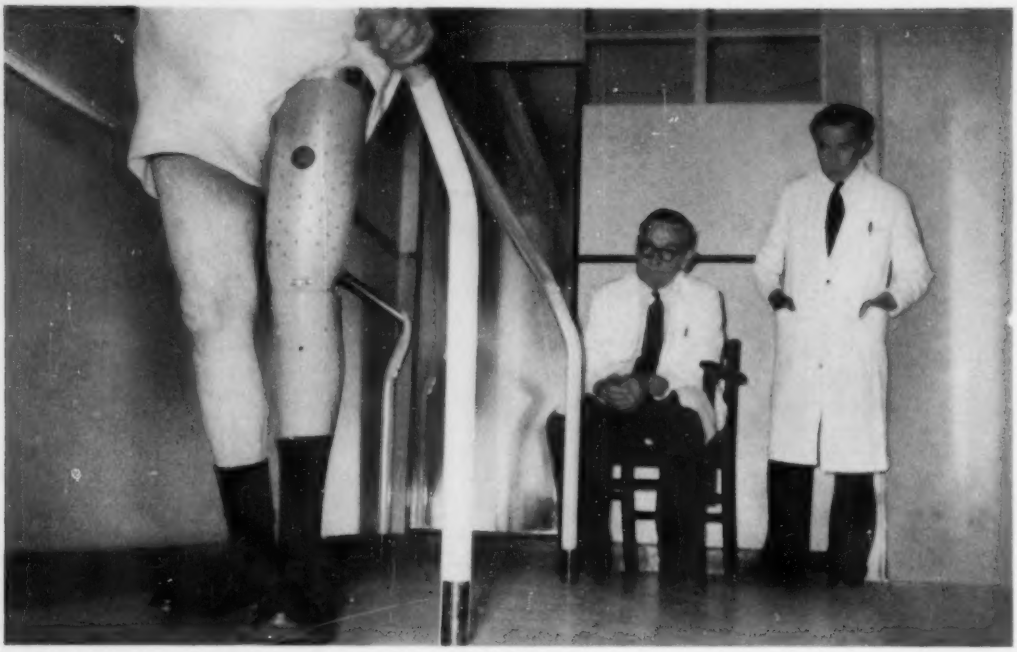

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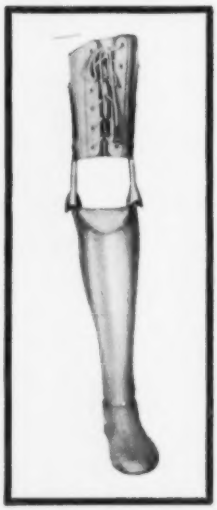

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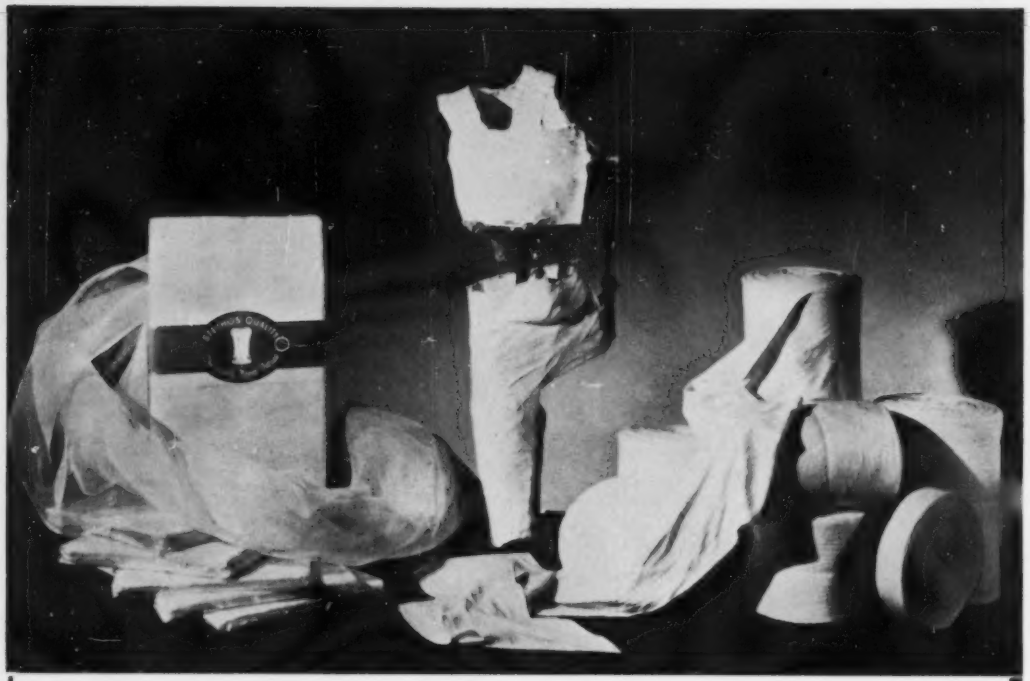

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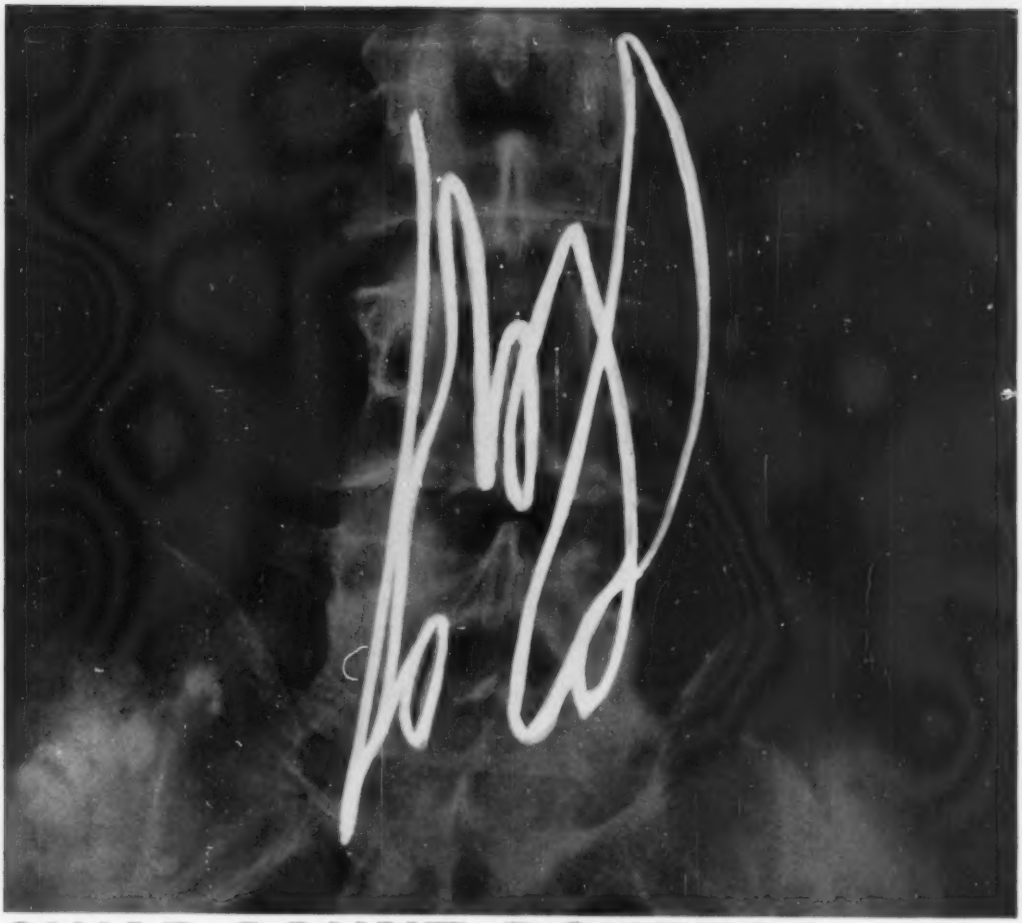

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Complete literature and references available upon request.

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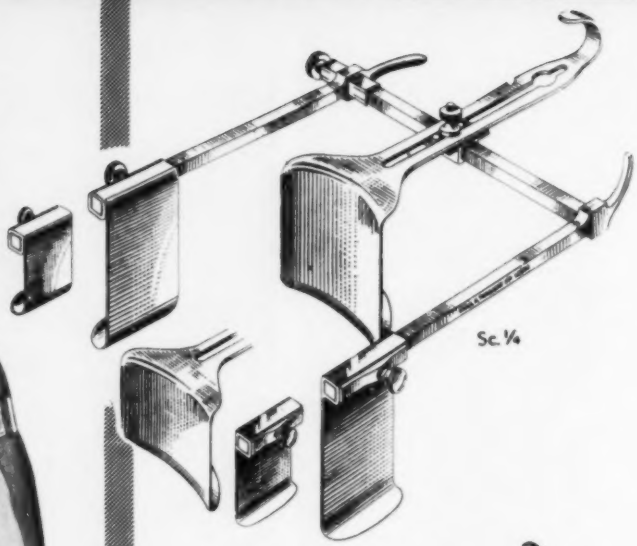

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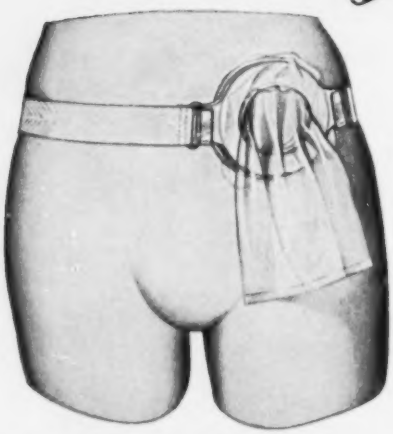

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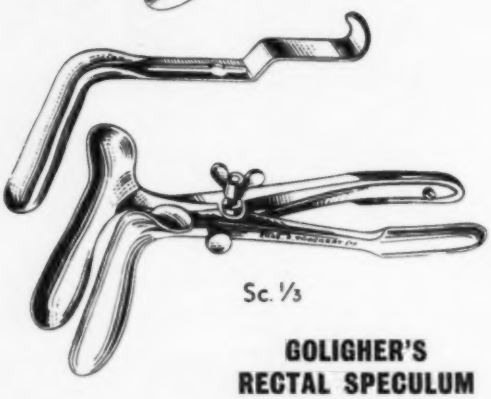

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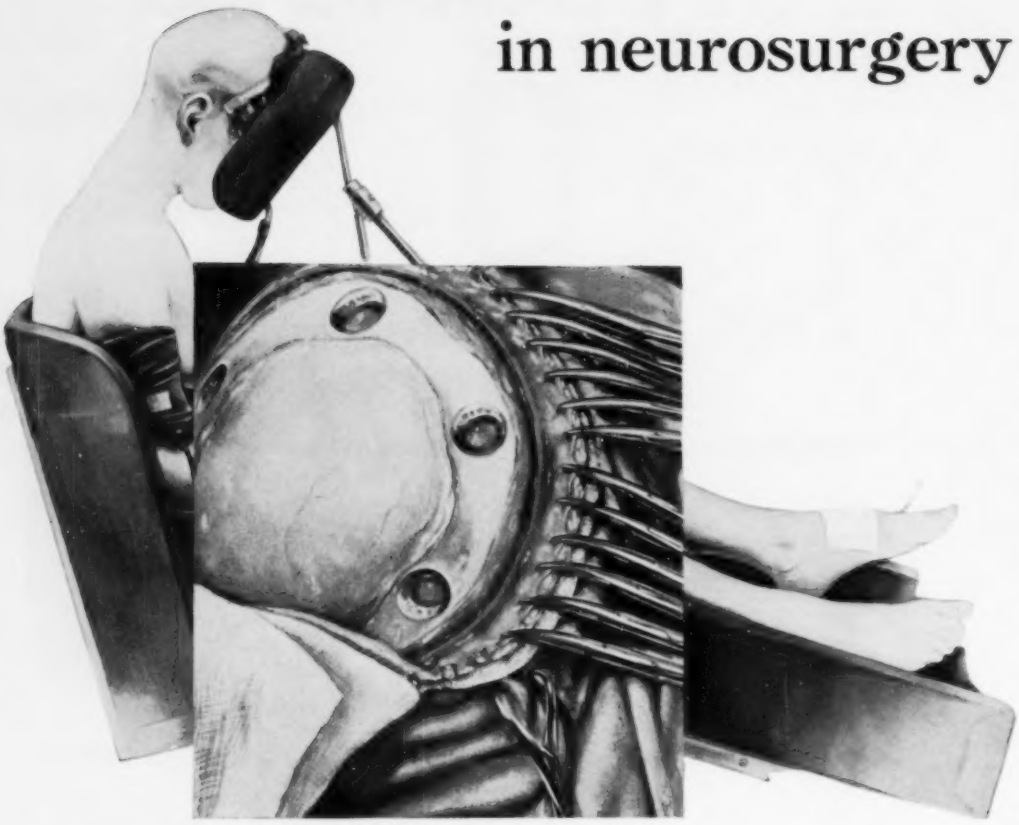

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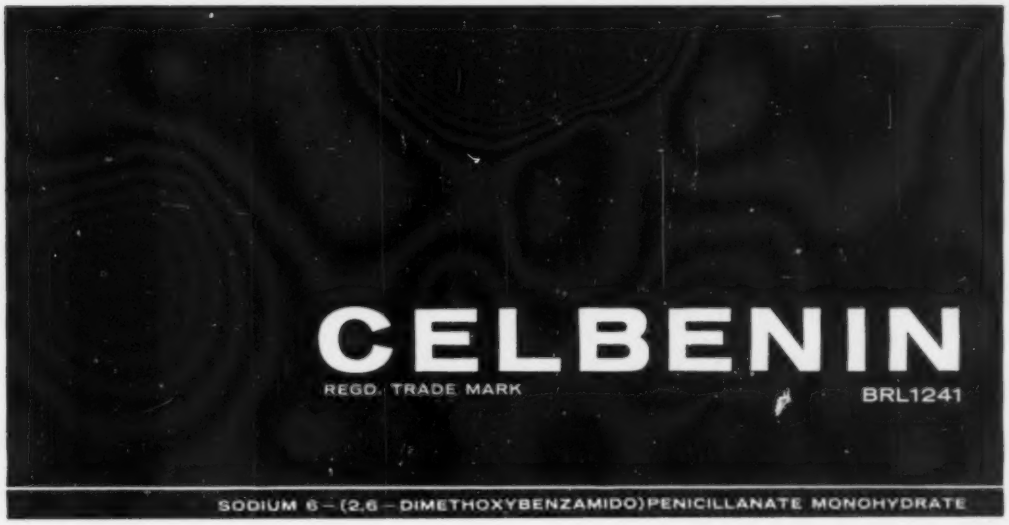

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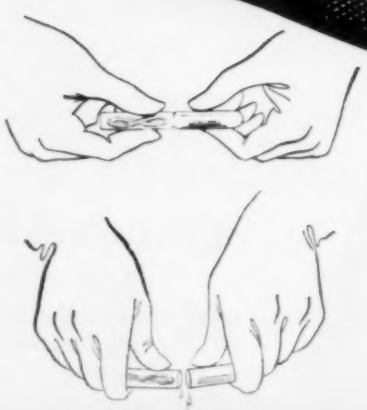

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VOL. XLVIII MAY, 1961 No. 212

In Memoriam

GEOFFREY JEFFERSON

An Appreciation

POSSIBLY the only person who could write this appreciation pleasingly and properly would have been Geoffrey himself. He would have ranged widely over biographical reminiscence and historical fact. Musingly he would have given freely of his personal assessments and impressions, deliberately planting here and there a little poetry in the guise of prose. Probably his pen was at its best when caricaturing the endearing qualities of his immediate and distinguished colleagues. But his acute insight would never have allowed him not to notice an imperfection, whether acquired or inherited, and so high-light it and time it and place it that over all there would emerge a faithful and engaging reproduction of the true personality he was wishing to portray. This facility for verbal portraiture was certainly part of his skill and possibly during his life called as much attention to his talents as anything else. The writer of a memory is apt to throw as much light on his own personality as on the one he is supposed to honour if he is not careful. If Geoffrey had one attribute it was perspicacity. Certainly he was fond of flattery, in fact he often said he liked it laid on with a trowel. Some of the praise that has been lavished on him recently would have been absorbed into his personality to enrich it, but he was highly selective and wisely discriminative and where truth was concerned always prepared to be rejective.

For obvious reasons it is in retrospect that most of his friends must view this complex man in his wisdoms, knowledge, and distinctions. But very much would be missed and the excitement of his living lost if an attempt were not made to go back with him into his formative years. He came to Salford shortly after the first world war. The 1920's for doctors were certainly exciting times and opportunities were limitless. Jeff's future was still in the melting pot. To the resident staff at the Salford Royal Hospital, to put it politely, he was enigmatic. He always came late and then bugled us about; explanations and certainly apologies were unknown. Soon a close study of him was made and our first stratagem met with some, but not complete, success. Warby, now of Wallingford, his first houseman, was put to bed at 4 o'clock in the afternoon, encouraged to sleep, and then was hurriedly awakened at Jeff's approach to make his afternoon rounds at 11 p.m. By this means we felt at least one of us would be ready and capable of carrying the battle into the early hours of the morning. Unfortunately for the rest of us, Jeff's presence was so attractive, and, indeed, so compelling that we also remained to the bitter end, finally going to bed half dead. At this period he was a strange character: very few understood him and some, because of his apparent determination to neglect his own interests, thought him feckless. However, in his colleagues he was unusually fortunate, for Garnett Wright, Jimmy Macalpine, Bob Ollerenshaw, and Ashton Smalley were by then not only specializing, but were already big and successful. They were rather older than Jeff and they not only accepted his foibles, but encouraged him and, indeed, at this critical period mothered him. If this had not been so, things might very easily have turned out differently. But in retrospect it seems more likely that this favourable attitude at Salford was merely an unfolding of a phase in destiny. Probably the only man at this period to whom Jeff made clear his high intentions was the late Robbie Galloway, the Resident Surgical Officer who later became a distinguished surgeon at the North Middlesex Hospital. He convinced Robbie; and all Robbie's prognostications of Jeff's academic triumphs came true. However, his final success surprised no one who knew him then.

Now having set the stage and erected the backcloth let us go to the beginning.

On one occasion, when travelling with him from the north-east into the Midlands, he had contrived to miss the direct train and we made our way south by the more circuitous coastal route. He was unusually silent and obviously preoccupied. As we passed through Stockton he became agitated, walked into the corridor, and beckoned me to follow. In the distance away from the sea he pointed to what looked like a small village and insistently asked where it was and what it was and what it did and why it was there. I didn't know the answers and in fact I am now quite sure he didn't want them. He knew them. This was the first time that I realized that he had had any personal associations with the County of Durham. When the penny dropped I quizzed him, but nothing came of it and I've made no further inquiries. But he would have done.

I have left these possible early associations with another county without further development because there is no doubt in essence that he was a full-blooded Lancastrian.

Rochdale was his home town and though unlikely for long to contain a man of Jefferson's stature,

there his choice of humour must have been conditioned. Occasionally he mentioned with nostalgic tenderness his recollections of his father's practice, of the Hornets, of Gracie Fields, and others. He was proud of his father's intellect, but it was from his mother that he inherited his distinctive habit of lateness. Of his brothers' and sisters' successes he was inclined to be whimsically reticent. He rarely spoke of his schoolboy days. None the less, it was at that ancient and distinguished seat of learning in the Long Millgate of Manchester that the basis of his classical scholarship was so firmly and broadly laid. And scholar above all else Jefferson certainly was.

Sir Geoffrey Jefferson, from the painting by Sir Gerald Kelly

From Lord James, the High Master of Manchester Grammar School, one of his dearest friends, I learn that Geoffrey Jefferson was a pupil on the classical side from 1899 to 1904. He continued to study the classical side until he reached Matriculation standard and then left for the University. There was nothing particularly distinguished about his school career, and he had little or no training in the basic sciences.

As a student at Owens College he had a most successful academic career, graduating with distinctions both in the University of Manchester and of London. His clinical training was carried out at the Royal Infirmary which became in every sense his Alma Mater. To no other institution did he show such devotion and such reverence. Professionally it was his spiritual home and there he remained happy and satisfied.

Now we come to the early 1930's when Jefferson was beginning to blossom in the professional sense, having by this time become firmly established at the Manchester Royal Infirmary. In this period every facet of his education was being taken care of by all around him, and it would be no exaggeration to say that his entourage was, if not anything else, virile. He had been profoundly stimulated, possibly overcome, by his visit to Russia during the 1914–18 war, and there is no doubt that he had been very tickled by his association with the Russian Royal family. Indeed, with the entourage the Royal visit had become a bit of a joke. One day after a long operating session, with the boys sitting around in the theatre suite, all still undressed as we would be long after the 'do' was over, the Master sat at the head of the table. He pulled out the famous Russian photograph with the crowned heads in the centre and Geoffrey on the periphery, and passed it round. It was bound to happen—there was a pause—a silence—and then it came in a drawl—"Which is the Tsar?" Words, of course, cannot precisely convey the nuance of the inflection of the voice in which this subtle impertinence was wrapped. As everyone now would expect there was no immediate reply, just a few sniffs and an odd puff on the ubiquitous cigarette. Then with a twinkle it came. "You weren't really in doubt—were you?" Voice matching voice.

Sir Gerald Kelly at work on the painting

It was in this iconoclastic school that Jeff had opportunity to practise and polish his repartee which later was to become so devastating.

Once I travelled with him into north Lancashire to operate on a patient in a large hall. In those days operating in private houses was quite common. We had had a long session and both of us were dehydrated, the electrolytes were in imbalance, and the CO_2 level high as the result of rebreathing and suffocating in the interest of asepsis. But that is by the way. When we were alone Jeff looked across laconically, proffering the information that someone in the next room was asking if we expected to be paid. I said "Of course we do! How are we going to get back?" He said, "That's right", but he still doubted if he could tell him. When I asked him why not, he said he would be afraid he would be asked "How much?" and would be unable to say. Finally it was decided that if we subtracted what we thought we would get from what we thought we were worth and divided it by two that would be about right. Coming back in the car he had obviously got a packet in his pocket,

but he never mentioned it. The next day he told me that everything had gone all right and asked me whether I thought that I should get anything. Of course I said "No" and of course the inevitable happened. Whenever we happened to pass one of my friends he would take me over and introduce me as the exceedingly fortunate young man who didn't need any money. There was never a dull moment.

I mention this anecdote to give me opportunity to satisfy the curiosity that Jeff's reaction to money so widely aroused. He needed a lot and liked to have it, but found the discipline and fag of having to make it exceedingly irksome. When conflict came he was always altruistic where money was concerned. He always regretted that he was not born rich.

As he was a man who has become a legend for his stories and repartees, to attempt to recount them is dangerous because taken out of their context they are apt to lose their piquancy. In the knowledge of this I will take the risk and recount one of Norman Whalley's favourites.

One of Jeff's irresistible charms was that he would draw you into whatever he was doing and encourage you to enjoy his interests and excitements. In particular he seemed to need an audience, of all things, when he was consulting in private. The technique was to stand around unobtrusively. On this particular day he was interviewing a wealthy woman from the Midlands whose complaint was that of atypical neuralgia of her face. Jeff, knowing the difficulties of this disorder, intervened very little in the discussion, but at last, as was inevitable, he was cornered and she demanded a remedy to relieve her distress. Knowing that almost any kind of medical therapy would do no good and that surgery would be likely to do harm he very hesitantly suggested that whenever the pain was particularly bad she should take a tablet or two of aspirin. This was like a red rag to a bull and my lady blurted out in disgust, "That will do no good". Jeff immediately detected the truth in this observation, his fingers extended and his forearms supinated, and he gently replied, "Then I advise you not to take any". Immediately I went out and related this to Betty Armistead, and a deep hoarse yell rent the firmament.

According to his audience he altered his tones, but the essential type of humour was always there. This was the Jefferson sauce, the *sauce piquante* with which he was apt to serve up his meat or to give alone when he thought the occasion worth no better. People have often wondered if there was more than one Jeff. I can say, beyond all doubt, there was not. He never changed; morning, noon, and night he was the same. In Jeff there was no coarseness or vulgarity. In his severe illnesses the quality of his personality stood out. To suggest he was perfect or entirely magnanimous would be incorrect. Certainly he never quarrelled unnecessarily or made an unnecessary enemy by a careless witticism. Should he feel, however, the danger of someone standing in his way he was as ruthless as the next, certainly more subtle, and possibly more determined. As his immediate colleagues will know, at times he was selfish, indeed, not infrequently and then unutterably.

By the middle of the 1930's he had been through the fettling shops of Salford and Manchester. And so that there should be no misunderstanding 'fettling' is an engineering term meaning that department of an engineering works where rough castings are taken to have their nobbly protuberances chipped off and made ready for finer polishing and precision fitting.

The second world war came at a time in his career that might easily have been catastrophic for a lesser man. He was not merely flexible—he was ready. Ready to unleash all that had been already stored up within him.

In his teachings his generosity knew no ends. Moreover, he was entirely catholic in his choice of audience. When he had tired everyone else out, doctors and nurses, he would start on the porters. On more than one occasion he stopped to make sure that the man on the lodge gate was fully cognizant of the difficulties he had just encountered and, of what means he had taken to put things right, and pictorially if necessary. He had the happy knack of making everyone not only interested, but participants in his activities.

In his out-patients he had favourites, and "Mrs. Osteo-encephalitis" often took tea with him.

He was immensely proud of the honours that showered upon him and he was very well aware how much of his public advancement and recognition he owed to his dear and life-long friend Harry Platt. Without Harry's distinction, maturity, and 'know-how' it is very unlikely that Jeff would have gone as far as he did. Again, here he was more than fortunate in his friendships.

For a while I will pass over my composition to another pen held by a member of the other sex, since there would be something lacking in a picture of Jeff that did not also depict his social glitter.

"To write only of the public honours and professional life of Sir Geoffrey would not convey to anyone who had never met him a particular quality of the man as he is remembered by those who knew him in his private life; the tall, rather heavy figure, the eyes half closed as he would stand musing for a moment before answering a question in that unique, soft, high-pitched, lazy voice, typical of all the Jeffersons.

"As a visitor in one's home his presence permeated the house. Everyone felt the need to please him in some special way, to remember some particular fancy—such as his taste for greengages. He assumed in some intangible manner the role of honorary great uncle to the family, and could sit and enjoy the exploits of 'Sooty' with the youngest member, shaking with subterranean mirth, as easily as he could listen seriously to the ambitions and enthusiasms of the eldest.

"Time became of no account; that he should be three hours late for dinner was as nothing, in a visitor who in the middle of his stay with you would suddenly decide to fly to Damascus and yet be back in time to see a game by his favourite football team—Sunderland.

"His charm, so difficult to capture in words, was probably dependent chiefly on the combination in a great man of humour and simplicity. There was a warmth in his personal contacts which makes his death a personal loss to all who knew him well."

If this attempted appreciation were merely to be a vignette of a vivid personality, cultured in humour

and successful in surgery, then not only would my task be now completed, but I believe I would never have been asked to make it. No one would have been sufficiently interested. Indeed, the legend that is Jeff might never have arisen and certainly would not have lived for long. Something more than a personality is needed if one's memory is to be stored in the temple of fame. His greatness which we are now honouring was, I believe, innate and owed nothing to his circumstances. He was completely occupied by a creative urge, and that he chose neurology was purely fortuitous, though the choice was felicitous. It was probably Grafton Elliot-Smith who influenced him most in these formative medical days. From his schoolboy days he knew where he was going and felt no anxiety about the ultimate outcome. Any peculiarity of behaviour was the outcome of the urge of his creative destiny. This urge allowed of no weakness, no distractions, and no concessions. This was the Jefferson whom only few in the early stages could know. From his Salford days he was carefully choosing the materials with which he was to build his life's work, constantly scrutinizing and arranging his shapes and colours with meticulous and loving care. This was his inner strength and he did not hesitate to use the magic of his personality as his smokescreen should his high intent be assailed either wittingly or unwittingly. He did not build in retrospect; he was far too wise for this; he accumulated his material and built as he went along. What was in the records was in his mind. It was the excitement of adduction that not only spurred him to further observation but quickened his receptiveness and prompted his questionings.

As a surgeon he was apt to boast that his writings on operative surgery were minimal; and for a good reason. In the brilliant Manchester school of Arthur Burgess, John Morley, Wilson Hey, and Billy Douglas he could be classified as no more than adequate, but he liked to operate and very much at odd times, like Sunday mornings and statutory holidays. Surgery of course enhanced his study of neurology, since as Sherrington once said, "It is nice to have a preparation that can answer back". He was meticulous where the welfare of his patients was concerned.

As an author he never wrote a long treatise and possibly for the good reason that this was not part of his *métier*. He was, on the other hand, a brilliant short-story writer. No one could make the problems of neurology so vivid and exciting. "I will make them listen" was his constant boast. In the use of the English language he was an artist. He spoke as well as he wrote, which is unusual. One of his most engaging skills was the way he used his asides or anecdotes as emphasis without tiresomely obscuring his argument or holding up the stream of his story. If you will read his compositions carefully enough you may detect a technique of phraseology that was not far removed from that of Wilfred Trotter's. He never divulged what this technique was, but it is none the less there.

And now to his wife Gertrude. She, in a different way, was his equal, and equally charming. She was completely occupied by her love for Geoffrey. As Shaw gave his name to 'Shavian' so Gertrude created the cult of 'Trudyism'.

It was on the solid foundations of his family life that Jeff was able safely to erect his creation. Without these solid foundations the edifice might easily by its own weight have sunk into the quicksands and disappeared, or have crumbled as the tremors of jealousies passed beneath it. He was too wise to use his wife as a spearhead to his designs and I doubt whether she would ever have consented. But, on the other hand, he never hesitated to throw her into his defences. Defences made up of a mixture of cream, wet blankets, and a willingness not to understand when necessary were formidable, indeed impenetrable.

And finally we come to, and cannot avoid, an objective prophecy of what posterity might think about him. That Jeff was good was immediately obvious to all; that he was better than good no one knew better than himself. But was he a genius? That he occasionally took himself to the borderlands of revelation and nibbled there must be accepted. That he did not cross the boundary and stay there was equally obvious. The sixty-four thousand dollar question you must answer for yourself. All I will say is that it is my opinion that the books of his *Collected Papers* will become more important as time passes and will be found well thumbed on the shelves of the scholars of future medicine. In that he created a masterpiece.

The life's blood of creative work is in the audience and its appraisement. Tragically the artist cannot create for himself alone, for his own delights, for his own satisfaction and fulfilment.

That Jeff was sensitive, the following excerpt from one of his letters will show: ". . . of your review of The Book I am as you can well imagine, deeply touched and enormously pleased! it would be stupid of me to pretend that I did not care—I do and what is more . . ."

Requiescat in pace.

GEOFFREY JEFFERSON

Born April 10, 1886. Elder son of Dr. Arthur J. Jefferson, a graduate with honours of London University who was in practice at Rochdale. Educated at Manchester Grammar School and Manchester University, which he entered in 1904.

Degrees and Distinctions:—

1907, Gold Medal in Anatomy at 2nd M.B. examination.
1909, Graduated with London M.B. with honours.
1911, Held Diploma of London Colleges, passed for the Fellowship of the Royal College of Surgeons.
1911, Resident at Manchester Royal Infirmary as House Surgeon, then to Tite Street as House Surgeon at Children's Hospital.
1911, Demonstrator in Anatomy.
1913, M.S. (Gold Medal).
1935, Professor of Neurological Surgery.
1943, *C.B.E.*
1947, F.R.S.
1950, Knighted.
1940 to death, Consultant Adviser in Neurological Surgery to the Ministry of Health.
1948–52, Member of the Medical Research Council.
1953–7, Chairman of newly created Clinical Research Board.

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By L. T. COTTON

KING'S COLLEGE HOSPITAL AND MEDICAL SCHOOL

LITTLE information about the anatomy of varicose veins can be found in the considerable literature which has accumulated on venous disease, and it is apparent that speculation about their origin has not been based upon direct observation. Previously it has been difficult to obtain suitable specimens of veins, but, now that the operation of vein stripping has made available an unlimited amount of material for examination, it is possible to describe the morbid anatomy of varicose veins and to relate this to appearances in life both at operation and in phlebograms. The purpose of this paper is to describe in detail the gross anatomy of varicose veins and to examine theories which are held about their development. An important feature of the investigation has been the preparation of plastic casts of veins, for it is only when these vessels are distended that the changes they have undergone can be fully appreciated.

MATERIAL

1. Normal Veins.—

a. Seventy-five specimens of the long saphenous vein have been removed from cadavers of which the legs showed no evidence of venous disease. The veins were removed by dissection from the level of the medial malleolus to and including the saphenofemoral junction.

b. In six limbs plastic casts of the long saphenous vein were prepared in situ (Appendix, 1); the casts were later removed from the limbs by dissection.

c. Plastic casts of the deep and superficial veins of six amputated limbs with normal veins were prepared (Appendix, 2).

2. Varicose Veins.—

a. Specimens of varicose long saphenous veins were obtained from stripping operations. The veins were either opened and examined (75 specimens) or they were reconstructed (27 specimens) and plastic casts of them were prepared (Appendix, 2).

b. When varicose veins were observed in the legs of post-mortem subjects (10 specimens) the long saphenous veins were removed from the level of the medial malleolus to the saphenofemoral junction, and, in addition, as much as possible of the femoral vein was removed. Plastic casts of the veins were then prepared (Appendix, 2).

c. Other methods of investigation included ascending phlebography (28 legs); examination of the saphenofemoral junction at operation (41 operations); and the preparation of plastic casts of varicose veins and deep veins found in amputated limbs (5 specimens).

RESULTS

1. Nomenclature.—The nomenclature of the venous valve advocated by Franklin (1937) has been used (*Fig.* 689). Valve cusps (E in *Fig.* 689) are diaphanous, tough membranes, crescentic in shape, with prominent cornua (D). The angles made by the attached borders (C) of the valve cusps where they meet is of some importance in the pathology of

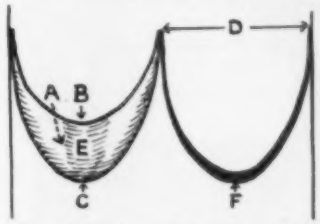

FIG. 689.—Nomenclature of the venous valve. (A, sinus; B, free border of valve; C, attached border of valve; D, cornua; E, valve cusp; F, agger.)

varicose veins and will be referred to as the 'cornual angle'. The 'agger' of the valve (F) represents the abrupt change in thickness of the wall of the vein which occurs where the valve cusp is attached; above the agger lies the thin-walled sinus of the valve (A). The free borders of the valve cusps (B) are in apposition in the normal vein.

The cornua of a valve lie very constantly in the transverse axis of the vein. The plane of the surface of a valve cusp relative to the long axis of a vein is more variable. It has been shown by Edwards (1936) that the surfaces of the cusps of a valve in a superficial vein lie parallel to a tangent drawn to the skin lying over it. In this position the competence of the valve is least likely to be disturbed by external pressure. Venous valves may be ostial or parietal, depending on their position. Ostial valves are sited at the actual entry point of a tributary into a larger vein; parietal valves are scattered along a vein at irregular intervals.

2. Varix Formation and Venous Valves.— When the valves of a varicose, long saphenous vein are examined large sacculations are frequently found which are constant in position in relation to the valves. These localized dilatations are referred to in this paper as 'varices' and are distinguished from the cylindrical dilatation also shown by varicose veins. Varices are best displayed in plastic casts. In 27 plastic casts of varying lengths of the varicose long saphenous vein there were 51 valves. Of these 31 showed a localized dilatation near to a valve and 16 appeared to be normal. No localized dilatations were found unrelated to valves.

Whenever a dilatation was found near a valve it always lay adjacent to the distal side of the valve cusps; that is on the upstream side of the valve. A normal valve has a dilatation, the sinus, immediately proximal to it (*Fig.* 690). The configuration of the sinus is preserved in varicose veins (*Fig.* 691) although it is usually enlarged. In all the plastic casts

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of varicose veins the varices lay immediately below the cornual angles of the valves (*Fig.* 691) and considerably increased the magnitude of the angles.

The varix is limited to one side of the valve (*Fig.* 691). The cornual angle of the valve on the side of the vein opposite the varix is only widened if the vein is greatly dilated; even then the asymmetry produced by the varix remains marked (*Fig.* 692). In some casts of varicose veins it is obvious that the varices are disposed along the length of the vein in an elongated spiral (*Fig.* 693). In others, the spiral arrangement is less apparent but, in these veins, varices project from alternate sides of the vein. It is probable that when the long saphenous vein becomes varicose it becomes twisted, the valves become rotated, and the varices share in the rotation.

FIG. 690.

FIG. 691.

FIG. 692.

FIG. 693.

FIG. 690.—A cast of a normal valve. The prominences formed by the sinus (S) are seen lying above the attached borders of the valve cusps, which have been marked with white ink. (× 2.)

FIG. 691.—Two views of a cast of a varicose vein taken at 90°. The varix projects from beneath one of the cornua of the valve. (× 1·5.)

FIG. 692.—A large varix showing dilatations below both cornua, one very much larger than the other.

FIG. 693.—A cast of a varicose long saphenous vein showing how varices may project spirally along the course of a vein. A thread has been attached to the varices to demonstrate their spiral appearance.

FIG. 694.—Plastic cast of a normal saphenofemoral junction. (F.V., femoral vein; L.S.V., long saphenous vein; T.V., terminal valve; S.T.V., subterminal valve.)

Examination of valves near the saphenofemoral junction at operation suggests that the twist on the vein ends below the femoral vein. Presumably the saphenofemoral junction acts as a fixed point which resists torsion. Direct inspection of varices at operation has confirmed that their appearance in casts is identical to that in life.

3. The Valves of Varicose Veins.—Two valves are constantly found in the terminal 5 cm. of the long saphenous vein (*Fig.* 694). One lies at the saphenofemoral junction; it will be known in this paper as the 'terminal valve'. The second lies within 5 cm. of the terminal valve and will be called the 'subterminal valve'. The terminal valve is an ostial valve; the subterminal valve is a parietal valve. In *Table I* is shown the incidence of terminal and subterminal valves in the long saphenous vein. It cannot be said that the presence or absence of either of these valves is in any way related to the development of varicose veins.

a. Terminal Valve.—The terminal valve of varicose veins can readily be inspected at operation.

If the long saphenous vein is divided 1 cm. below the saphenofemoral junction the cusps of the terminal valve can be seen within the lumen of the proximal stump of the vein. In 22 operations the terminal valve was examined; in 14 the valve cusps were obscured by a reflux of blood through the valve, but in 8 there was no reflux and the valve cusps were normal in appearance. The valves which were found competent at operation were not necessarily competent under normal conditions, for stimulation of a vein by dissection causes it to contract vigorously and this contraction could easily render an otherwise incompetent valve competent. Observations made at operation confirm that incompetence of the terminal valve is not due to any disease of the valve but is caused by dilatation of the long saphenous vein.

Table I.—The Incidence of Valves in the Terminal 5 cm. of the Long Saphenous Vein

(T.V., terminal valve; S.T.V., subterminal valve.) In varicose veins the presence or absence of the terminal valve was noted at operation.

No. of Veins	No. T.V.	Per-centage T.V.	No. S.T.V.	Per-centage S.T.V.
53 Normal	50	94·3	52	98·1
38 Varicose	36	94·7	36	94·7

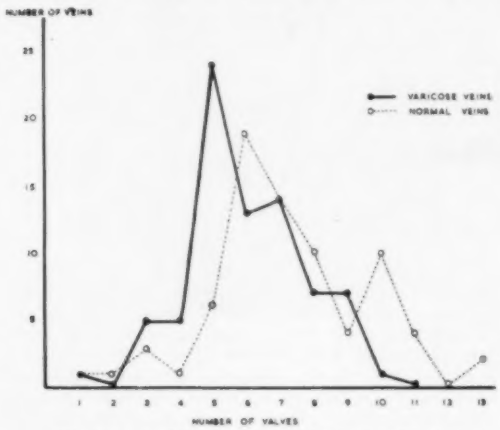

Fig. 695.—A histogram of the number of valves in 75 varicose and 75 normal long saphenous veins (exclusive of the terminal valve). There are fewer valves in varicose veins.

b. Valves distal to the Saphenofemoral Junction.—Distal to the saphenofemoral junction the valves of the long saphenous vein are very variable in number. In *Fig.* 695 is shown a histogram of the number of valves in 75 cadaver veins and in 75 stripped veins from medial malleolus to saphenofemoral junction but exclusive of the terminal valve. The difference in the mean number of valves in normal veins ($7{\cdot}3 \pm 2{\cdot}3$) and in varicose veins ($6{\cdot}0 \pm 1{\cdot}7$) is statistically significant (p less than 0·001).

It can be shown that the smaller number of valves in varicose veins is not due to a primary deficiency of valves but is the acquired result of venous dilatation. If a vein is distended with air or gelatin before fixation by formalin it can readily be demonstrated that an increase in the girth of a vein puts tension on its valve cusps (*Fig.* 696). The valve cusps of a dilated vein remain joined at their cornua and are unchanged in size, although their shape is altered. Only when a vein is distended can it be seen how the valve cusps are tensed and made to project like narrow flanges into the lumen of the vein. The strain on the valves produced by pathological dilatation leads to their sclerosis and, eventually, to their disappearance.

The majority of valves in stripped varicose veins appear to the naked eye to be normal. For example, of 156 valves which were examined, 123 were macroscopically normal; only 33 were sclerosed. The sclerosis was often so marked that only the rim of the valve was left at its attached border. Sclerotic changes in the valves were never seen in normal cadaveric veins.

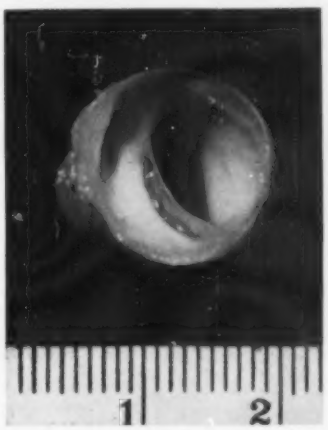

Fig. 696.—A distended varicose vein cut transversely through the sinus to show how the valve cusps are tensed by distension and project into the lumen.

c. The Distribution of Valves and Varices along the Long Saphenous Vein.—In *Fig.* 697 is shown the situation of valves along the course of both normal and varicose, long saphenous veins. Along the normal vein, below the subterminal valve, the valves are randomly scattered. In the varicose vein the larger part of its deficiency in valves lies in the proximal two-thirds of the vein where it is most dilated (*see* 4, *below*). A similar deficit appears to exist near the ankle (*see Fig.* 697), but this is probably not real, for the lower end of the long saphenous vein is often so damaged at operation that valves are destroyed and hence not recorded.

Varices are not found beneath all the valves in the varicose long saphenous vein; normal valves often intervene between those which exhibit varices. The commonest position for a varix is below the subterminal valve (*Fig.* 697). Only very rarely is a varix found below the terminal valve (present in only 3 of 41 operations). Varices are most obvious in the proximal part of the vein where it is cylindrically dilated. In the lower leg varices are either absent or

so small as only to be seen in injected specimens (*Fig.* 698). These small varices occur in veins which are either undilated or are only a little dilated.

The 'saphena varix' which is so often seen in the groin is usually the varix below the subterminal valve. It is most obvious when the vein is greatly enlarged; when the subterminal valve lies close to the terminal valve; and when the varix lies on the anterior surface of the vein. Varices are not invariably found below the subterminal valve. In 9 of 41 operations there were no varices below either the terminal or subterminal valves and yet there were prominent varicose veins farther down the leg. In plastic casts of the varicose long saphenous vein it is not uncommon to find the largest of varices in the centre of the vein.

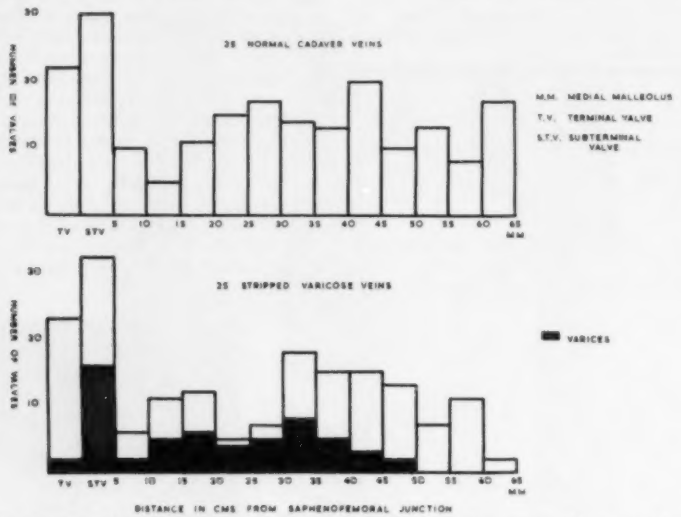
25 NORMAL CADAVER VEINS
NUMBER OF VALVES
M.M. MEDIAL MALLEOLUS
T.V. TERMINAL VALVE
S.T.V. SUBTERMINAL VALVE
TV STV 5 10 15 20 25 30 35 40 45 50 55 60 65 MM
25 STRIPPED VARICOSE VEINS
NUMBER OF VARICES
VARICES
DISTANCE IN CMS. FROM SAPHENOFEMORAL JUNCTION

FIG. 697.—The situation of valves in normal and varicose long saphenous veins. The positions of varices have been recorded in varicose veins.

FIG. 698.—A cast of a relatively undilated part of the varicose long saphenous vein. Three valves are shown (1, 2, 3), the middle of which has a small varix below it. The upper and lower valves are unaffected. (× ½.)

d. Ageing and Valves.—It has been claimed (Bardeleben, 1880) that the number of valves in the normal long saphenous vein diminishes with age. This view has not been confirmed in a survey which was undertaken of veins from cadavers of known ages at death. Fifty long saphenous veins were examined from medial malleolus to saphenofemoral junction. The mean number of valves (7·0) in 23 veins at ages from 17 to 59 years was not significantly different from the mean number of valves (7·2) in 27 veins at ages from 60 to 90 years. The terminal valve was not included in counting the number of valves.

4. The Course and Calibre of the Varicose Long Saphenous Vein.—The clinical appearance of legs affected by varicose veins gives the impression that the long saphenous vein is itself tortuous. However, the tortuosity is due to varicose tributaries which overlie the main trunk of the vein and only appear to be part of it (*Fig.* 699). The relation of the tributaries to the main trunk can be demonstrated at operation when a vein stripper is found to pass unimpeded from the ankle to the groin deep to intensely contorted tributaries. In plastic casts of varicose veins the only deviations from the normal direct course of the long saphenous vein are the bulges produced by varices (*Fig.* 700). The varicose long saphenous vein shows some lengthening, but the lengthening is slight compared with that undergone by tributaries of the vein (*see* **5**, *below*).

The varicose long saphenous vein is cylindrically dilated in that part of the vein which runs through the upper part of the lower leg and through the thigh. In the lower leg the vein is often normal in calibre or only a little enlarged. The change in calibre of the

vein is often abrupt and coincides with the point of entry of a large varicose tributary (*Fig.* 701); at this point it is not uncommon to find the largest tributary in the long saphenous vein. In normal veins there is a similar change in calibre as the vein approaches the knee, but the change is very small.

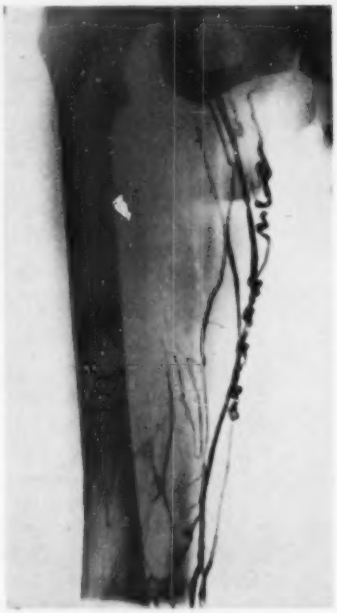

FIG. 699.—A phlebogram showing tortuous tributaries overlying a dilated but relatively straight long saphenous vein.

5. Varicose Tributaries.—Tributaries of the varicose long saphenous vein become greatly elongated, tortuous, or even spirally wound (*Fig.* 702). At first they exhibit little increase in diameter, but later they become both cylindrically dilated and sacculated (*Fig.* 703). Unlike the varices in the main trunk of the long saphenous vein, these sacculations bear no relation to valves.

Varicose tributaries may appear anywhere along the saphenous vein. The commonest site of their early appearance is below and near to the knee. It is rare for a varicosity to develop in the named tributaries which enter the saphenous vein near its junction with the femoral vein. These tributaries are often enlarged, but they rarely become tortuous or sacculated unless the femoral vein has been the site of previous thrombosis or unless an operation has previously been performed on veins in the groin.

Varicose tributaries often show signs of recent or old thrombosis. The extreme tortuosity of these vessels must lead to stasis of blood within them and this stasis may predispose to thrombosis. It is, however, rare to find thrombosis in stripped veins; presumably the flow of blood in the long saphenous vein is faster and more direct than in its varicose tributaries, and hence the tendency to thrombosis is less.

6. Communicating Veins.—Communicating veins have been studied in preparations of plastic casts of normal and varicose veins in amputated lower legs (*Fig.* 704). Communicating veins are very variable in size and length; some are paired and form venæ comitantes of a cutaneous artery. Most contain valves, and the function of those without valves appears to be preserved by valves in the deep and superficial veins which lie near to the communicating veins.

Varicose communicating veins show changes similar to those found in other tributaries of the long saphenous vein. The communicating veins become longer and moderately tortuous. The most extreme degree of tortuosity is exhibited by small tributaries which are found close to and draining into communicating veins (*Fig.* 704).

FIG. 700.

FIG. 701.

FIG. 700.—A cast of the proximal two-thirds of a varicose long saphenous vein and femoral vein. The vein runs a straight course to the groin. (× ½.)

FIG. 701.—A large tributary (T) entering the long saphenous vein (L.S.V.) just below the knee. At the point of entry of the varicose tributary the long saphenous vein is greatly dilated. Below this level the vein is undilated. (× ½.)

7. Deep Veins.—The deep veins have been examined in phlebograms of 28 limbs affected by primary varicose veins. In all the phlebograms the popliteal and femoral veins were found to be of normal calibre and to contain normal valves. The contours of the deep veins of the lower leg were also unchanged, but, as the valves of these veins cannot usually be seen in phlebograms, their positions could only be inferred from the outlines of the valve sinuses. When varicose veins of cadavers have been removed for the preparation of plastic casts (10 specimens) the femoral veins have been inspected, and in none has any abnormality of the valves or any change in the wall been found.

Plastic casts of the deep veins of the lower leg of limbs which showed varicose veins (6 specimens) have been compared with the deep veins of legs which showed normal superficial veins (6 specimens). There were no abnormalities in the posterior tibial,

peroneal, or popliteal veins which could be related to the presence of varicose veins. The contours and valve spacing of the deep veins of limbs with normal superficial veins could not be distinguished from that of limbs with varicose veins.

When plastic casts of varicose superficial veins are traced through the communicating veins to the deep venæ comitantes it is obvious that the varicosity stops abruptly at the junction of the communicating veins and the deep veins. The veins of the calf muscles are not easily examined, for neither in phlebograms nor in plastic casts can they be completely filled. However, in none of the phlebograms examined nor in the

FIG. 702.—Varicose tributaries in an injected amputated limb. The veins are greatly elongated but relatively little dilated. (× ⅓.)

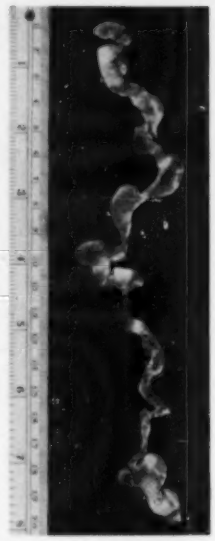

FIG. 703.—A cast of a varicose tributary at a late stage of development. There are saccular dilatations which project from alternate sides of the vein.

FIG. 704.—A, a normal and B, a varicose communicating vein. The intensely tortuous veins which are seen in B are varicose tributaries entering the communicating vein. (C.V., communicating vein; L.S.V., long saphenous vein; P.T.V., posterior tibial veins.)

plastic casts of veins of amputated limbs has there been any evidence of varicosity of the deep veins, either venæ comitantes or muscular veins. In one plastic cast one of the gastrocnemial veins was dilated, but this was not thought to be an example of deep varicosity.

DISCUSSION

1. The Varix.—The most interesting feature of varicose veins is the position of the varix in relation to the venous valve. In such a common disorder it is surprising that the correct position of the varix has not been noted more often. It has been said to lie proximal to the valve (Callender, 1862; Rindfleisch, 1867; Bennett, 1890); proximal ('sinusectasia') or distal ('true varices') to the valve (Lowenstein, 1908); and to have no fixed relation to the valve (Schwarz, 1934). By many other authors it has been inferred, though perhaps not stated, that the varix lay proximal to the valve. Trendelenburg (1891), Slawinski (1899), and Hasebroek (1916a, b) have described the varix in its correct position immediately below the valve. The reason for the failure of so many authors to recognize the true site of the varix is that it is only when varicose veins are distended that their varices stand out. When veins are opened before examination their varices are collapsed and easily missed.

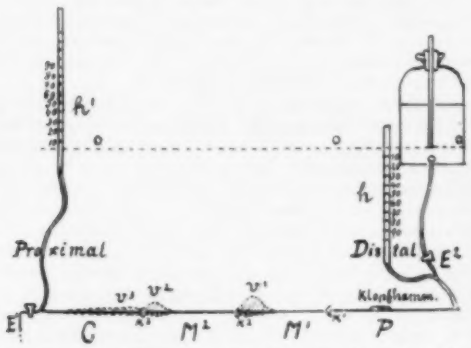

FIG. 705.—Illustration taken from Hasebroek (1916a), showing the method of artificial production of varices by means of pulsatile flow in an elastic, valved tube.

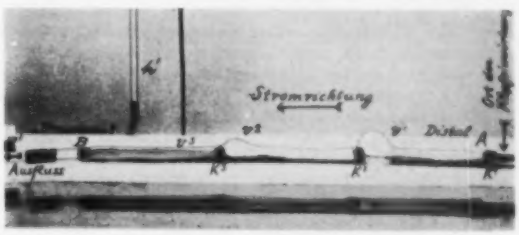

FIG. 706.—Photograph taken from Hasebroek (1916a), showing the appearance of artificially produced varices. There is a marked similarity to the appearance of varices in plastic casts.

It was suggested by Trendelenburg (1891) that the venous valve normally protects the wall of the vein below the valve from the pressure of the blood in the vein. According to this view, if the terminal valve of the long saphenous vein should give way, the part of the vein below the valve dilates because it is not built to withstand the full impact of the hydrostatic pressure of a column of blood from the heart to the groin. Trendelenburg's view presupposes that the venous valves serve to divide up veins into segments containing blood at low pressure. In fact the resting venous pressure throughout both normal and varicose veins is the hydrostatic pressure exerted by a column of blood from the right auricle to the point of measurement. During exercise, the pressure in normal veins falls markedly below the hydrostatic level. In varicose veins exercise produces a much smaller effect, the pressure falling only slightly or not at all (Hojensgaard and Stürup, 1952).

In the early stages of development of varicose veins it is most uncommon to find a varix below the terminal valve even though lower down the leg, for example below the subterminal valve, large varices may be present. The absence of a varix below the terminal valve is difficult to explain, but it does suggest that the development of varicose veins is not solely due to the effect of an increased pressure upon part of the vein unaccustomed to bear it. No anatomical evidence has been brought forward to substantiate the view that the vein is weak below the valve. It can easily be confirmed by naked-eye examination that the weakest part of the vein is the sinus above the valve where it would be expected to give way if a heightened hydrostatic pressure were the sole cause of varicosity.

If a valve becomes incompetent it is possible for a regurgitant stream of blood to impinge upon the wall of the vein below the valve and there weaken it from the effects of turbulent flow. The experiments performed by Holman (1955) in the investigation of 'post-stenotic dilatation' have shown clearly the weakening effect that turbulent flow has upon the arterial wall. However, as valve cusps are attached transversely at right angles to the long axis of veins, it might be expected that, if regurgitation of blood were the cause of the varix, the dilatation should affect the whole circumference of the vein. The striking feature of the varix is its asymmetry, and this asymmetry cannot be explained by regurgitation through an incompetent valve.

Large varices appear only in that part of the vein which is cylindrically dilated—that is, in the proximal two-thirds of the vein. It is possible that pathological dilatation of a vein in some way disturbs the architecture of the vein wall immediately below the valve. Confirmation of this view must await a detailed examination of the microscopical structure of the vein wall in relation to the valve.

Venous dilatation puts tension upon the cusps of the valves. This effect has been clearly described by Rokitansky (1852): "The valves increase to a certain degree with the dilatation of the vein but, after a time, they cease to increase and are no longer capable of closing the enlarged vessel; they then lie in a state of tension transversely across the tube of the vein." The tension on the valve cusps causes them to project into the lumen of the vein as rigid flanges. It is possible that the rigidity of valves in dilated veins may alter conditions of blood-flow on the distal side of the cusps. Ledderhose (1906) suggested that there normally exists an increased pressure, stasis, and

turbulent blood-flow immediately below a valve. These factors were said by him to be increased when a vein became diseased or dilated, for the valve ring was relatively rigid and failed to increase in diameter as much as the vein adjacent to the valve. However, the appearance of plastic casts of varices does not support Ledderhose's suggestion of a relative narrowing at the valve attachment.

The formation of the varix was studied by Hasebroek (1916a, b), who set up a model of a vein in the form of a very thin-walled latex tube in which had been inserted three single-flap, non-return valves (*Fig.* 705). Water was passed through the tube at pressures comparable with those found in veins. The flow was made pulsatile by a clockwork hammer which could be made to tap regularly on the side of the tube at different frequencies. At certain critical frequencies of pulsation 'blow-outs' appeared upstream of the valves (*Fig.* 706). Hasebroek interpreted his experiments in relation to the cause of varicose veins by suggesting that pulsation, slight in degree, could occur in superficial veins. The pulse had been communicated through communicating veins from the venæ comitantes of the leg.

The experiments of Hasebroek have been repeated by the author and Dr. J. M. Zarek in the Department of Engineering at King's College, London. Exact replicas of Hasebroek's model vein were constructed and repeated attempts were made to produce dilatations upstream of the valves by a pulsating flow of water. Dilatations could be made to appear if the stream were considerably narrowed at the sites of the valves and also at points of weakness, due to unevenness in the thickness of the latex tubing. However, there was no evidence of a critical combination of pressure, flow, or frequency of pulsation which would regularly produce blow-outs proximal to the valves. It is theoretically possible that pulse-waves might be reflected at the valves and cause small pressure disturbances or turbulent flow which might lead to the development of dilatations below the valves, but this possibility could not be confirmed by means of the model.

As yet the problem of the origin of the varix is unsolved. In the opinion of the author, two possibilities must seriously be considered. First, the varix may arise from a structural change in the wall consequent upon venous dilatation; and, secondly, despite our failure to reproduce Hasebroek's results, the varix may arise from some abnormality of blood-flow. An examination of the architecture of veins is at present being undertaken to help elucidate this problem.

2. The Valves of Varicose Veins.—The venous valves have often been blamed for the appearance of varicose veins. Terms commonly used to describe the abnormal valves include 'weakness', 'inborn deficiency', and 'primary valvular deficiency'. Valves have been held to shrink with age (Klotz, 1887) and with the vein wall to suffer from the effects of metabolites in the blood (Zancani, 1911). The common appearance of varicose veins in athletes has been attributed to the rupture of venous valves by exercise (Homans, 1916). It was claimed by Bardeleben (1880) that the number of valves in the long saphenous vein decreased with advancing age, the largest number of valves being found in the fœtus.

The series of normal and varicose veins which has been examined by the author has not produced evidence to substantiate any of the diverse views that have been expressed concerning the lesions affecting the valves of varicose veins. There are clearly fewer valves in varicose veins than in normal veins, but the reduction in the number of valves is unrelated to any effect of ageing. Valve cusps disappear from the effects of prolonged increased tension produced by venous dilatation. This increased tension produces sclerosis and contraction of the valve and eventually its disappearance. An opposite conclusion was reached by Edwards and Edwards (1940), who, from serial sections of veins, described separation and slackening of the valve cusps following venous dilatation. They did not comment upon the position of the varix below the valve or how the cusps remained joined at their cornua no matter how much the valve attachment was lengthened.

3. The Course of the Varicose Long Saphenous Vein and its Tributaries.—A striking feature of varicose veins is the difference between the changes undergone by tributaries of the long saphenous vein compared with the main trunk. The intense tortuosity and elongation of the varicose tributary is comparable with the tortuosity of collateral vessels around a site of arterial obstruction. The great lengthening of venous tributaries can be likened to the effect of prolonged stress upon an elastic structure. The stress is at first resisted, but later fatigue occurs and the structure loses its elastic properties. If the stress continues the material becomes greatly elongated and fails to return to its original length when the tension upon it is released.

The absence of a similar degree of elongation of the main trunk of the long saphenous vein may possibly be related to the greater thickness and strength of that vessel. It is also possible that the structure of the tributaries may differ from that of the main trunk. Variation in the pattern of muscle, collagen, and elastic fibres may lead to different reactions to stress.

4. The Deep Veins.—Evidence for the occurrence of deep varicose veins dates from the observations of Verneuil (1855). However, the large veins which Verneuil described in the calf were found by Delbet (1921) to be present normally in the soleus muscle. To Le Dentu (1867) can be attributed the first full descriptions of the normal anatomy of the deep veins of the leg. First, there are long channels which run the length of a muscle; for example, in the gastrocnemius muscle each head is drained by a pair of long thin venæ comitantes draining into the popliteal vein. Secondly, short anastomosing arcades of veins run through the length of a muscle and enter the posterior tibial and peroneal veins at regular intervals; this pattern is to be seen in the long flexor muscles. The soleus muscle exhibits both patterns; long channels run the length of the muscle and are connected to the posterior and peroneal veins by short arcades. The accuracy of the observations made by Le Dentu has been confirmed by Dodd and Cockett (1956), who described large veins in the normal soleus muscle in the form of large sinuses which are unvalved. These are the veins which have in the past been mistaken for deep varicose veins.

It is difficult to examine the deep veins of the leg, for neither by phlebography nor by injection of amputated legs is it possible to fill all the deep veins. However, examination of phlebograms and plastic casts of limbs bearing varicose veins shows convincingly that varicosity does not occur in the posterior tibial, peroneal, popliteal, or femoral veins. Of the few specimens of muscular veins that have been available for examination none has provided evidence of varicosity in the muscles of the calf. When communicating veins have been examined it has been most striking how varicosity ceases abruptly at the junction of communicating and deep veins. These facts lead to the conclusion that deep varicose veins do not occur. The same conclusion was reached by Olivier (1950) and Mathiesen (1959) after examination of large series of phlebograms.

CONCLUSIONS

It has been the purpose of the author to describe the changes to be found in varicose veins and to examine some of the views which have been held in the past as to their origin. The results of the investigation have been useful in excluding many factors which have been said to be causes of varicose veins. Two conclusions can be drawn from this survey. First, primary varicosity is a disease of superficial and communicating veins; the deep veins are not affected. Secondly, the lesions of the valves in varicose veins arise following venous dilatation; they do not precede it.

The origin of varicose veins cannot be attributed to the development of a descending valvular incompetence originating at the saphenofemoral junction. Some unknown factor causes superficial veins of the leg to dilate. Whether the cause of the dilatation is endocrine, hæmodynamic, or some inherent structural weakness in the vein has yet to be ascertained.

SUMMARY

The gross anatomy of the normal and varicose long saphenous vein has been investigated in fresh specimens obtained from cadavers, stripping operations, and amputated legs. Some of the results derived from these methods of investigation have been confirmed at operation and by phlebography.

Near the saphenofemoral junction two valves have been described, the 'terminal valve' and the 'subterminal valve'; it is below the latter that the 'saphena varix' usually arises. The number of valves in the varicose long saphenous vein is smaller than in the normal vein and the deficit is due to venous dilatation. No evidence of a primary valvular disease has been found. Increasing age is not accompanied by disappearance of valves and phlebitis rarely occurs in the long saphenous vein though it is common in its tributaries.

Prominent dilatations occur in the varicose long saphenous vein immediately below the cornua of the valve cusps. These varices are unilateral and are often arranged spirally along the length of the vein. They are most obvious in the cylindrically dilated part of the vein (that is, in its proximal two-thirds), except below the terminal valve where a varix is rarely seen. The origin of the varix has been discussed.

The varicose long saphenous vein shows little lengthening, whereas its tributaries are greatly elongated, tortuous, and even spirally wound. When fully developed, varicose tributaries show sacculations which project from alternate sides of the vein. Communicating veins are affected similarly and the tributaries which drain into them show extreme tortuosity.

No evidence has been found of varicosity of the deep veins of the leg so far as they can be examined. The difficulty of investigating the muscular veins of the lower leg has been stressed.

APPENDIX

1. The Preparation of Plastic Casts of Cadaver Veins in Situ.—The long saphenous vein is cannulated at the level of the medial malleolus and the external iliac vein in the groin. The long saphenous vein is then washed free of blood and clot with tapwater followed by acetone. 'Vinylite' resin, made by dissolving VYHH powder (Bakelite Ltd.) in acetone to form a 10 per cent solution, is injected into the vein at the ankle until the iliac vein is distended. After two hours the plastic is hard enough to allow the superficial veins to be removed by dissection. The specimen is allowed to dry, after which it is then possible to remove the wall of the vein by peeling it off the cast.

2. Preparation of Plastic Casts of Veins after Removal from the Body.—Stripped veins are prepared by closure of all holes and tributaries by ligature—a most laborious process. Amputated legs are prepared by cannulation of the long and short saphenous veins and the posterior tibial veins at the ankle and the popliteal vein near the knee. The veins are washed through with tapwater to remove blood and clot. Varicose veins found in cadavers at post-mortem examination are excised through a long incision extending from the ankle to the groin. A wide margin of subcutaneous fat around the veins is also removed to include as many tributaries as possible. The veins are washed through and any leaks closed by ligature.

The plastic used for injection is Marco resin 26C (Scott Bader & Co. Ltd.), made up as follows:—

Resin 20 g.; Monomer 10 g.; Catalyst H 1·6 g.; Accelerator E 0·9 ml.

A small amount of blue pigment to colour the cast to the desired degree.

This mixture sets hard after 20–40 min. Twenty-four hours after setting, the soft tissues around the cast are removed by maceration with commercial concentrated hydrochloric acid.

Acknowledgements.—This work was carried out with the aid of a grant from the Research Committee of King's College Hospital and Medical School, to whom I wish to express my gratitude. I am indebted to Springer-Verlag, of Heidelberg, for permission to reproduce *Figs.* 705 and 706.

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ANTISEPSIS OF SURGEONS' HANDS

A STUDY OF VARIOUS AGENTS UNDER THEATRE CONDITIONS

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THE incidence of post-operative wound sepsis has been an increasing source of worry in recent years, particularly since large numbers of antibiotic-resistant staphylococci have come to be so widely distributed in the hospital environment. Any method, no matter how remote, that may reduce the risk of introducing bacteria into clean surgical wounds should be thoroughly evaluated.

The incidence of glove puncture in the operating theatre was assessed by Devenish and Miles (1939) to be as high as 24 per cent.

The use of a modern electric device for detecting puncture (Penikett and Gorrill, 1958) has produced results indicating that the percentage of faulty gloves may be as high as 30 per cent. In view of these findings it is obvious that organisms present on the surgeon's hands frequently must pass through the gloves into the tissues of the patients.

Considerable experimental work has been carried out to establish the efficiency of various skin antiseptics in relation to wound infection. In 1944 Traub, Newhall, and Fuller showed a marked decrease in skin flora following the daily use of soap containing the substance G.11 (hexachlorophene). In 1947 Seastone published experiments demonstrating the advantages of hexachlorophene as a skin antiseptic. This was followed in 1948 by the work of Hufnagel, Walter, and Howard, who successfully combined 3 per cent hexachlorophene with an anionic detergent cream, Phisoderm. Their experiments with this cream ('pHisoHex'*) proved it to be much superior to other antiseptic techniques tested in the reduction of skin flora. In 1955 Murray and Calman showed that the use of chlorhexidine cream in obstetric practice decreased the incidence of cross-infection. This was followed by the work of Smylie, Webster, and Bruce (1959) comparing the use of pHisoHex with the standard operating scrubbing technique and showing a marked reduction in skin contaminants. Recently Lowbury and Lilly (1960) compared six different scrubbing methods under laboratory conditions, using amongst other things substances incorporating chlorhexidine and hexachlorophene. We have assessed the relative efficiency of various skin antiseptics under operating theatre conditions in respect of their antiseptic qualities, their speed of action, and the duration of their effect.

* pHisoHex is manufactured by Bayer Products, The Winthrop Group, Surbiton, Surrey.

MATERIALS AND METHODS

Six surgeons co-operated in these experiments; they are identified alphabetically in the experiments to be described. Apart from emergencies, these surgeons operated three times a week on Monday, Wednesday, and Friday. The relative action of the antiseptics used was demonstrated by the following technique: impressions of the palmar and dorsal surfaces of the fingertips of the operating hand (the dominant hand) were taken after scrubbing (prior to putting on gloves) and again post-operatively immediately after removal of the gloves. The impressions were taken onto 5 per cent horse blood-agar plates which were subsequently incubated overnight at 37° C. The bacterial counts were then recorded for both palmar and dorsal surfaces independently by counting the number of bacterial colonies appearing on each finger impression. The duration of each operation was recorded in minutes and this time was taken as an indication of the time between finger cultures. Initially, to act as a control, the standard hospital scrubbing technique was investigated for 2 weeks. This method consists of a 5-min. scrub with a sterile nylon nailbrush and bar soap over the whole skin surfaces of the hands and arms as far as the elbow, followed by a tapwater rinse and a 95 per

cent alcohol rinse. The hands were dried on a sterile towel. After this initial control period, 5 different antiseptics were incorporated into the scrubbing technique in turn, for similar lengths of time, with an interval of 3–4 days between each new trial period. The substances used were: (1) pHisoHex (3 per cent hexachlorophene suspended in an anionic detergent cream Phisoderm); (2) Soap containing 2 per cent hexachlorophene; (3) Dettol antiseptic cream;* (4) Hibitane (chlorhexidine) obstetric cream;† (5) Chlorhexidine (½ per cent) in 95 per cent alcohol. The scrubbing method used in the case of pHisoHex was a 4-min. technique as follows: (1) take approximately 2 ml. pHisoHex into the palm of one hand; rub into the hands and arms to just above the elbow for 1 min.; (2) rinse well under running water; (3) clean the nails with a sterile nylon brush and pHisoHex under running water; (4) repeat stage (1) for 2 min., covering skin area as far as the elbow; (5) rinse well under running water; (6) dry on a sterile towel. The routine 5-min. scrubbing technique was used for all the other compounds, but the 95 per cent alcohol rinse was omitted, and in the cases of (3), (4), and (5) the application of Dettol cream, Hibitane cream, and chlorhexidine (½ per cent) in 95 per cent alcohol was substituted.

* Dettol antiseptic cream is manufactured by Reckitt & Sons Ltd., Hull.

† Hibitane obstetric cream is manufactured by Imperial Chemical Industries Ltd., Wilmslow, Cheshire.

RESULTS

The number of bacteria in each finger impression culture was calculated by the following arbitrary system: (*a*) −, sterile impression; (*b*) +, impression showing 1–15 colonies; (*c*) ++, impression showing 15–30 colonies; (*d*) +++, impression showing 30–45 colonies; (*e*) ++++, impression showing over 45 colonies including semiconfluent or confluent growth.

The results of the experiments are shown in *Table I*. The figures in this table represent the average numbers of + for the whole volar or dorsal surface (all five fingers) of each operator over the whole of the two-weeks' test period for each antiseptic tested. The maximum figure possible would be 20 (an operator whose finger impressions all gave confluent growths of bacteria throughout the test period) and the minimum 0.

It should be noted that the bacterial flora encountered during these experiments consisted of the following organisms: *Staphylococcus albus*, Micrococci, and, on one occasion only in each instance, *Bacterium coli* and *Staphylococcus pyogenes* (bacteriophage type 52/80). It will be seen from *Table I* that, using the standard scrubbing method, the post-operative counts are higher (except in the case of Surgeon B) than the pre-operative ones. It is also obvious that the pre-operative count for each surgeon varies considerably and that the dorsum of the fingers carries more bacteria than the palmar surfaces. In the case of the pHisoHex trial, not only were the

Table I.—RESULTS OF EXPERIMENTS

Standard Method				Surgeon	Hexachlorophene Cream			
Pre-operative		*Post-operative*			*Pre-operative*		*Post-operative*	
Volar	Dorsal	Volar	Dorsal		Volar	Dorsal	Volar	Dorsal
2·6	6·8	5·7	8·5	A	1·5	1·6	0·5	0·4
10·6	14·0	5·6	6·3	B	1·7	1·1	0·0	0·0
2·3	5·4	3·0	7·0	C	1·1	1·1	0·3	0·7
0·2	1·4	4·2	7·6	D	1·3	2·2	0·9	0·4
11·6	13·2	14·3	13·3	E	4·0	5·6	0·8	0·1
3·5	10·3	9·1	10·3	F	3·6	4·4	1·7	0·6
2 Per Cent Hexachlorophene Soap					½ Per Cent Chlorhexidine in 95 Per Cent Alcohol			
Pre-operative		*Post-operative*			*Pre-operative*		*Post-operative*	
Volar	Dorsal	Volar	Dorsal		Volar	Dorsal	Volar	Dorsal
5·8	8·6	5·8	6·8	A	8·0	6·0	8·5	3·2
2·0	1·0	0·7	0·8	B	8·0	6·0	6·0	6·0
0·0	0·1	0·5	0·3	C	7·0	6·0	5·0	8·0
2·6	10·0	6·5	6·3	D	1·5	3·5	2·2	4·0
7·0	6·0	3·0	2·4	E	12·0	8·5	12·0	9·5
5·6	11·3	7·2	8·3	F	4·0	4·0	2·0	2·0
Dettol Cream					Hibitane Obstetric Cream*			
Pre-operative		*Post-operative*			*Pre-operative*		*Post-operative*	
Volar	Dorsal	Volar	Dorsal		Volar	Dorsal	Volar	Dorsal
10·5	11·7	16·0	13·0	A	3·5	7·5	6·0	10·0
9·0	15·0	8·0	8·0	B	3·2	4·5	4·6	4·0
8·3	8·3	7·3	10·0	C	8·8	11·6	12·0	10·0
15·0	15·0	15·0	15·0	D	6·0	7·0	10·6	9·3
8·5	7·0	10·5	6·0	E	—	—	—	—
3·6	3·0	3·6	3·0	F	—	—	—	—

For explanation of figures see 'Results'. * Only 4 surgeons took part in this trial.

bacterial counts generally lower than the control group, but the post-operative counts were less than their pre-operative equivalents. With this substance the variation in pre-operative counts for each individual surgeon was greatly reduced and it is apparent that there was not such a marked discrepancy between the counts of the dorsal and palmar surfaces of the fingers when pHisoHex was used. We noted that during the course of these experiments the maximum antibacterial activity of pHisoHex was attained 30–45 min. after application and that this activity was maintained for at least 3 hr. This effect was not observed with the other agents examined, all of which showed their maximum bacterial effect almost immediately after application and then progressively declined as time passed.

Table II.—To show the Cumulative Antiseptic Effect of pHisoHex as compared with Standard Alcoholic Rinse

Surgeon	Before		After	
	Palmar	Dorsal	Palmar	Dorsal
A	2·5 (2·0) 1·0 (4·0)	2·5 (5·5) 0·5 (7·5)	0·3 (9·0) 0·5 (6·5)	0·3 (10·0) 0·0 (14·0)
B	7·0 (7·5) 0·0 (13·0)	4·5 (12·0) 0·5 (12·0)	1·0 (3·5) 0·0 (9·0)	0·5 (8·5) 0·0 (5·0)
C	1·75 (0·25) 0·5 (2·3)	1·75 (2·2) 0·0 (6·6)	1·0 (1·0) 0·0 (5·0)	0·75 (2·5) 0·0 (6·6)
D	2·5 (1·0) 0·5 (0)	8·5 (2·5) 0·5 (2·0)	0·0 (4·0) 0·0 (5·0)	1·0 (3·5) 0·0 (11·0)
E	1·0 (15·0) 2·5 (15·0)	2·0 (10·0) 3·5 (15·0)	1·0 (14·0) 1·5 (15·0)	0·0 (12·5) 0·5 (14·0)

The first figures given represent pHisoHex-treated hands and those in brackets alcohol-treated hands. In the case of each surgeon the top row of figures represents the bacteria found at the commencement of the investigation and the bottom row those found at the end of the investigation. Figures represent bacterial counts of finger impressions calculated according to the system explained in the text.

The manufacturers claimed that pHisoHex had a cumulative effect in that repeated use over a period would result in increasing bacteria-free skin. *Table II* shows a comparison of the operators' hands at the start and at the end of the experimental periods for the standard technique and for pHisoHex. It will be seen that there are undoubtedly fewer organisms on the operators' hands in 5 of the 6 surgeons examined after 2 weeks' regular use of pHisoHex.

DISCUSSION

It must be stressed that all the observations recorded were carried out under normal operating theatre conditions during the normal day-to-day hospital routine. As a result of this there was considerable variation beyond our control of certain factors. For example, some lists consisted of a series of short operations while others were made up of two or three lengthy procedures. This type of variation meant that the number of applications of any antiseptic was liable to considerable variation during any operating session. However, the results are considered to offer a reasonably reliable comparison of the substances used, as in all cases the number and duration of the surgical procedures approximate to each other over the whole of the test periods.

We concluded from the results that pHisoHex was the only substance tested that produced any real diminution of the numbers of bacteria on the fingers. It is noteworthy that hexachlorophene soap, while undoubtedly producing some effect on the number of bacteria, is not nearly so effective in this respect as pHisoHex either immediately after application or post-operatively. We consider that this is due to the fact that the method of application of the two agents differs in one important aspect: pHisoHex cream is applied to the dry skin of the hands and forearms at the start of the appropriate scrub and rinsing with water follows this application. Hexachlorophene is virtually insoluble in water and the application of water to skin smeared with pHisoHex causes the antiseptic to be precipitated onto the skin as a thin layer over all the treated area. This film of hexachlorophene is very tenacious and difficult to rub off. In the case of hexachlorophene soap it is necessary to wet the soap before applying it. Here the water causes precipitation of the antiseptic onto the cake of soap and not the skin. The skin has the already precipitated chemical rubbed onto it from the wet cake of soap and obviously the distribution will be patchy and poor, with the highest concentration on the palms of the hands and the bristles of the nail brush.

We consider that the two hexachlorophene compounds are superior to the other agents tested because the precipitate they form with water clings tenaciously to the skin, and very little of it is rubbed off on the sterile towel used for drying the hands before putting on gloves.

SUMMARY

With the co-operation of six surgeons, a series of experiments comparing antiseptics in surgical scrub techniques was carried out in the operating theatres of the Middlesex Hospital over a period of 5 months.

For each test period of 2 weeks a different antiseptic was used and an evaluation of the relative efficiency of each agent was made.

It was found that of the five substances described only one, hexachlorophene, reduced the skin bacteria significantly in relation to the standard scrubbing method evaluated as a control.

Further, the substance is shown to be most effective when employed as a cream (pHisoHex). A possible explanation for this finding is put forward.

We should like to thank those members of the Surgical Staff of the Middlesex Hospital who took part in the trial.

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UNILATERAL ABSENCE OF THE TESTIS*

EIGHT CASES OF TRUE MONORCHISM

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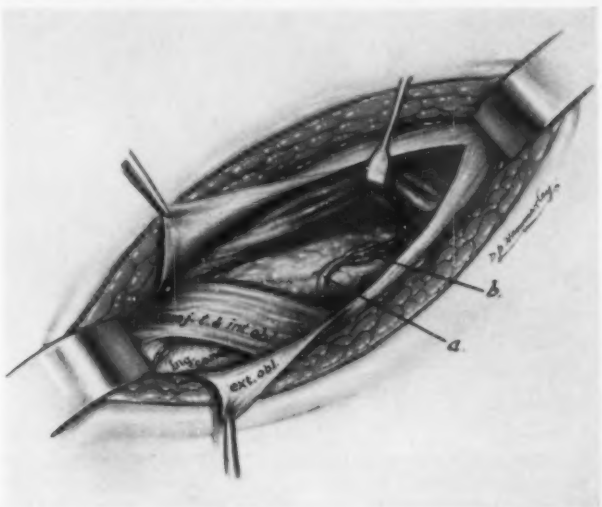
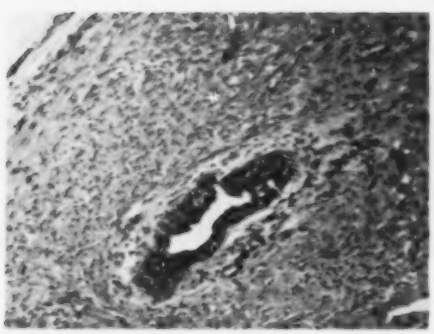
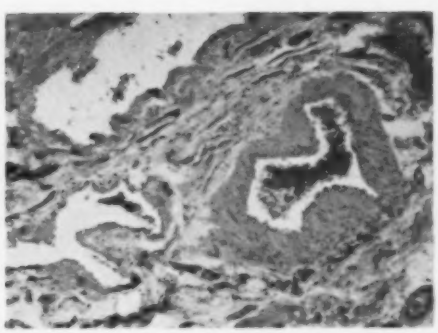
THE state of monorchism, due to complete failure of development of one testis, is well recognized but comparatively few cases are described in the literature. In 1949 Kawaiche, Cooper, and O'Donnell reviewed the 47 cases so far described and presented 2 more of their own. Marble (1956) and Williams and Lee (1956) described 2 further cases. Gross (1953) reported 24 cases of unilateral absence of the testis amongst 722 patients coming to surgery because of abnormalities of testicular descent; this suggests an incidence of 3 per cent amongst those cases of undescended testis likely to require surgery. Unilateral cryptorchism due to retention of the testis within the abdomen occurs with at least equal frequency and must be clearly distinguished from true monorchism.





a



b

FIG. 707.—Complete absence of the testis. Showing the method of surgical approach by muscle-splitting incisions giving access to the inguinal canal and the extraperitoneal tissues of the iliac fossa. The vas ends blindly in the iliac fossa and at its termination is joined by a leash of spermatic vessels. The histological appearances of the principal structures are shown. *a*, Vas deferens (× 61); *b*, Spermatic veins and artery (× 61).

The distinction between concealment and complete absence is of importance because, whereas the latter state is probably harmless, there is good evidence that the abdominal testis is sufficiently prone to malignant change to make its recognition and removal from the abdominal cavity most

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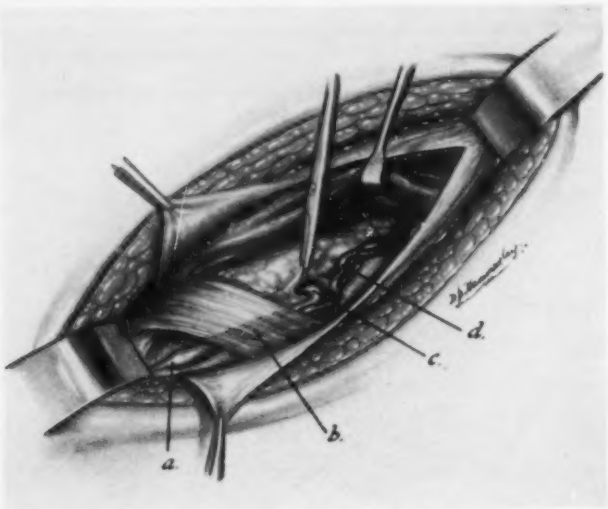
desirable, even if this entails orchidectomy in the event of failure to obtain a satisfactory scrotal position (Rea, 1931; Gordon-Taylor and Till, 1938; Gilbert, 1941; Bennett-Jones and Harrison, 1941). Experience in the north of England lends support to this, because amongst 91 consecutive cases of malignant testis on the records of the Registration Bureau of the Newcastle upon Tyne Regional Cancer



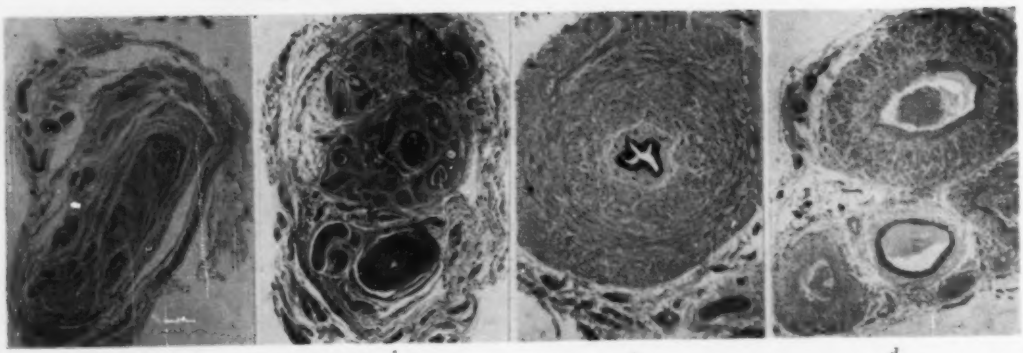


FIG. 708.—The same surgical approach as *Fig.* 707 has been used. The vas ends as a small bulb in the inguinal canal; it is accompanied by spermatic vessels descending from the posterior abdominal wall. Representative histological samples of the principal structures are also shown. *a*, The terminal bulb consists of fibromuscular tissue rich in blood-vessels (× 8). *b*, Vas deferens and associated spermatic vessels (× 11·2). *c*, *d*, The same structures at a higher level (× 28·8 and × 28).

Service for the years 1954–1959, 5 arose in inguinal ectopic testes in adults, 2 in abdominal testes, also in adults, and 2 in testes which had been transposed to the scrotum in adult life a few years previously. If it is accepted that 0·2 per cent of men have an undescended testis (Bishop, 1945), then these figures indicate that the undescended testis is fifty times more prone to malignant change than the normally descended testis. Gilbert and Hamilton (1940) arrived at an almost identical figure. The findings from the north of England may be used to suggest that the adult patient with any form of undescended testis is exposed to a risk in the order of 1 in 66 of developing malignant change in his lifetime (*see* Appendix). Malignant change in an abdominal testis is particularly dangerous because the condition is likely to declare itself only when it is far advanced. In the bilateral case this risk may have to be accepted (although this can be debated), but the fact that the danger can be avoided when a normal opposite testis permits excision of the abnormal side makes it particularly important that the boy with an abdominal testis should be correctly diagnosed and appropriately dealt with. The avoidance of a possible torsion and the removal of a coexistent hernial sac are incidental but significant advantages.

Until laparotomy, however, it is usually impossible to distinguish the abdominal testis from failed development of the testis; both states present clinically with an 'absence' of a detectable testis on one or both sides. If the state is bilateral, the patient may well be a problem in sex diagnosis, such as those

examples of pseudohermaphrodites described by Armstrong (1953, 1955, 1958) or of gonadal dysgenesis (Turner's syndrome) described by Hoffenberg and Jackson (1957), and a correct diagnosis may be

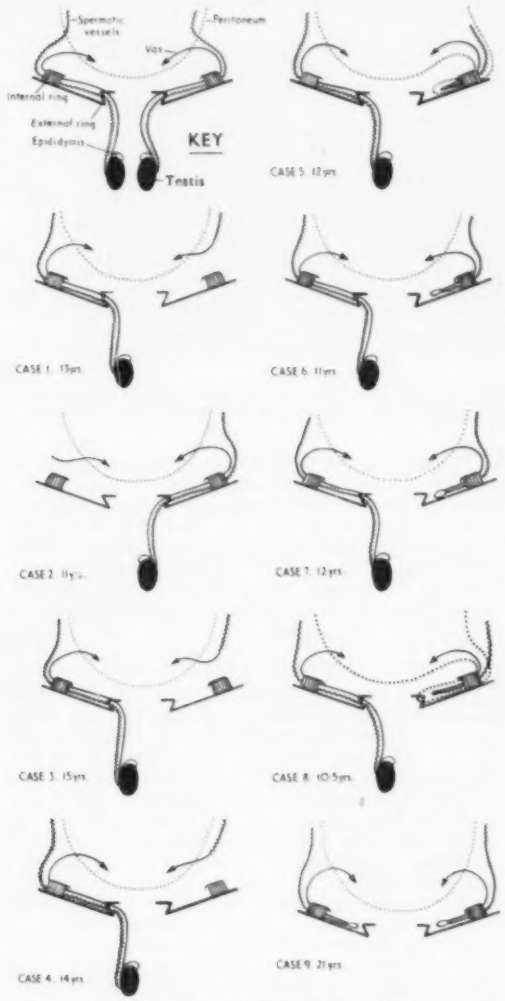


FIG. 709.—Diagrams summarizing the findings in the cases of absent testis.

possible on clinical grounds; moreover the surgeon, under these circumstances, will usually be prepared to use a midline transperitoneal exposure. In the unilateral state the surgeon should be able to obtain positive evidence of the true state by use of an incision which will permit easy exposure of the inguinal canal if necessary and which offers the minimal surgical interference. Failure to locate a testis may leave the surgeon wondering whether he has missed a small mobile testis retained within the abdomen or whether there has indeed been a complete failure of its development; to conclude incorrectly that there is no testis present is to defeat the very purpose of the operation.

This article is concerned with the means by which the surgeon may demonstrate conclusively in the patient with unilateral clinical 'absence' of the testis whether the missing testis is retained within the abdomen or whether there is a true complete absence of the testis on that side.

MATERIAL AND METHODS

Eighteen patients were explored surgically because no testis could be detected on one side on clinical examination. The ages ranged from 9 to 15 years. One other patient was explored because of bilateral 'absence' of the testes in an otherwise normal young man. Three further patients in whom there was bilaterally no exterior evidence of the testes were explored in order to solve a problem of sex diagnosis—1 of these was a pseudohermaphrodite with testicular feminism (Armstrong, 1953, 1955) and 2 were examples of Turner's syndrome; it is not proposed to consider these 3 patients here.

All 18 unilateral cases were explored by an incision separating the fibres of the obliquus externus aponeurosis over the inguinal canal and well laterally; this gave access to the canal and also gave ample room for the iliac fossa to be entered by splitting the obliquus internus and transversus muscles (*Figs.* 707, 708); this approach was extraperitoneal unless there was difficulty in locating the testis and then the peritoneum was opened to admit a finger to assist the search. If the testis was not easily located, attention was directed towards finding the vas deferens by appropriate extraperitoneal dissection; in 3 cases the vas deferens was located by palpation of the pelvic wall by a finger in the peritoneal cavity.

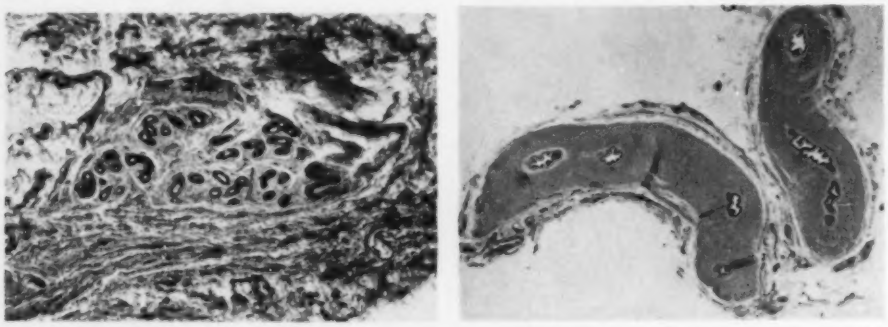
RESULTS

In 8 patients there was a complete absence of the testis on one side and in 1 patient there was bilateral absence of the testes. (The details of these patients are summarized diagrammatically in *Fig.* 709.)

In each case of unilateral absence the vas deferens was located without difficulty after a brief initial search failed to reveal a testis; the vas was traced either to a blind ending or to a small protuberance of tissue; in all cases but one (*Case* 2), there was a cluster of vessels running down to the termination of the vas deferens and this had the characteristic appearance and origin of the spermatic vessels. In 4 cases the vas ended subperitoneally in the iliac fossa (*Cases* 1–4 and *Fig.* 707); in the other cases the vas deferens ran into the inguinal canal and ended as a small bulb (*Cases* 5–8 and *Fig.* 708). In no case was tissue resembling a testis seen; in 1 case (*Case* 5) there was a hernial sac, in another case (*Case* 8) the processus vaginalis had persisted in a loculated form, and in every case the opposite testis was well developed and normally placed in the scrotum. The terminal portion of the vas deferens was removed and its identity confirmed histologically; in 2 cases tissue resembling the epididymis or vasa efferentia was identified; no histological evidence of the presence of any testicular tissue was found in any

case. Some typical examples of the histological findings are presented in *Figs.* 707, 708, 710, 711.

The patient with bilateral absence of the testes (*Case* 9) was found to have a symmetrical failure of testicular development; the vas ended in the inguinal canal as a slender expansion resembling epididymis, but no testis was present. Because this case was bilateral, no tissue was removed for microscopy. A leash of spermatic vessels ran down to the termination of the vas and the tissue thought to be epididymis; no hernial sac was present.



A B

FIG. 710.—The histological appearances of tube systems found widely distributed in the tissue around the hernial sac removed from *Case* 5. A, The tubules are lined with columnar epithelium and give every appearance of corresponding to vasa efferentia or the epididymis (× 25). B, A portion of the associated vas deferens is also shown (× 8).

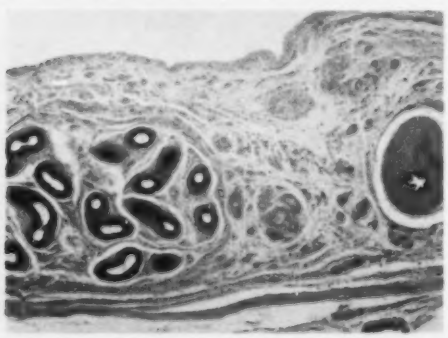


FIG. 711.—Part of the terminal bulb in *Case* 8. The vas is seen alongside a group of tubules with the characteristic appearance of epididymis (× 31).

In each of the remaining 10 patients an abdominal testis was located; all of these testes were easily recognized as such and were at least 1 cm. in diameter. (The details of these patients are summarized diagrammatically in *Fig.* 712.) On three occasions the testes eluded location until the vas deferens was identified and traced upwards. Five testes (*Cases* A, C, D–F) lay suspended within the abdominal cavity at the level of the pelvic brim, and 4 (*Cases* B, G, H, J) lay close to the internal inguinal ring; these 4 were associated with hernial sacs extending into but not beyond the inguinal canal. In the remaining case (*Case* I) a prolonged search failed to reveal any trace of a vas; the testis, apparently without epididymis or vas, was found protruding from the lower lumbar peritoneum into a recess beneath a high cæcum; a hernial sac was present in the inguinal canal. Histological study, however, revealed that a strand of tissue leading away obliquely from the testis contained a tube system resembling the epididymis (*Fig.* 713). In 6 patients the opposite testis was in a normal scrotal position and in the remaining 4 (*Cases* A–C, G) the opposite testis was or had been in the superficial inguinal ectopic position and associated with a hernial sac. It was possible to bring the abdominal testis to the upper scrotum in 3 patients in whom the gonad lay near the internal ring (*Cases* B, G, J); orchidectomy was performed in the other 7 patients, all of whom had an opposite testis satisfactorily positioned, either naturally or surgically, in the scrotum.

DISCUSSION

The observations presented above suggest that the vas deferens is likely to be present even if the testis has failed to develop; this is understandable because it arises from an independent structure. Kawaiche and others (1949), Marble (1956), Williams and Lee (1956), and Counseller, Nichols, and Smith (1940) were able to demonstrate a vas ending blindly in each of the 10 cases they describe. Thorek and Thorek (1933), quoting Sebileau and Descomps (1916), stress that there is always some remnant of the deferent canal in anorchism. In most, if not all, cases, therefore, the vas deferens forms a sure guide to the surgeon and enables him to conduct his search for the abdominal testis or for evidence of its absence with accuracy and subtlety through a small incision. (Emphasis of the value of this method is given by the fact that one of our abdominal testes had previously eluded the search for it by another surgeon who had concluded that no testis was present on that side.) Final confirmation of the true state is provided by the characteristic leash of spermatic vessels running to the termination of the vas deferens in cases of failed testicular development. The presence of these vessels possibly rules out the existence at a separate site of a testis which has not linked up with its epididymis or vas (the mesonephric components). Complete separation of testis from the epididymis and vas is possible; Kawaiche and others (1949) consider the available evidence on this (Counseller

and Walker, 1933; Thorek and Thorek, 1933; Lazarus and Marks, 1947) and it is not necessary for us to review this again; they conclude that separation is very unlikely to cause confusion. In the few

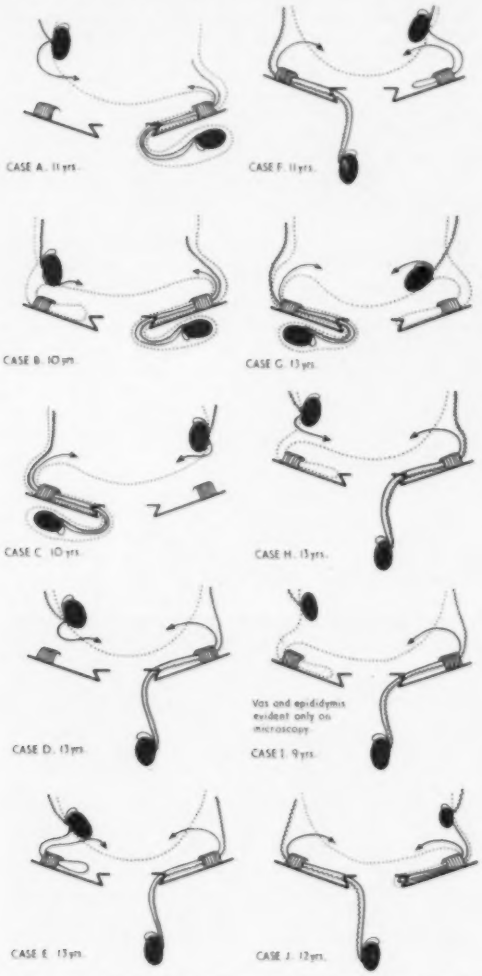

CASE A. 11 yrs.
CASE F. 11 yrs.
CASE B. 10 yrs.
CASE G. 13 yrs.
CASE C. 10 yrs.
CASE H. 13 yrs.
CASE D. 13 yrs.
Vas and epididymis evident only on microscopy
CASE I. 9 yrs.
CASE E. 13 yrs.
CASE J. 12 yrs.

FIG. 712.—Diagrams summarizing the findings in the cases of abdominal testis. The key is in *Fig.* 709.

reported cases of separation the testis has been found near the internal ring and this area was inspected sufficiently carefully in our own cases to make it improbable that a separate testis was overlooked.

The example of an abdominal testis without apparent epididymis or vas is interesting, because unless histological study had been made of all tissues leading to the testis it would have been concluded that the epididymis and vas were totally absent; in fact, on microscopical examination, the epididymal tube system appeared to connect with the seminiferous tubules (*Fig.* 713). It is possible that a vas also existed but was undetected because of similar poor development and the unusual course it must have followed to reach the testis. This possibility was not overlooked at operation and all likely strands seen in the pelvic dissection were biopsied without success. This case is regarded as an example of imperfect development of epididymis and vas deferens. The testis had its characteristic leash of

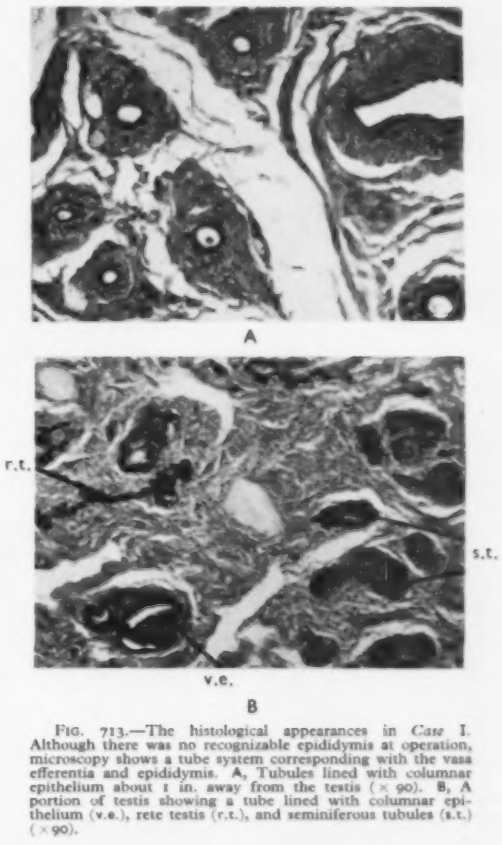
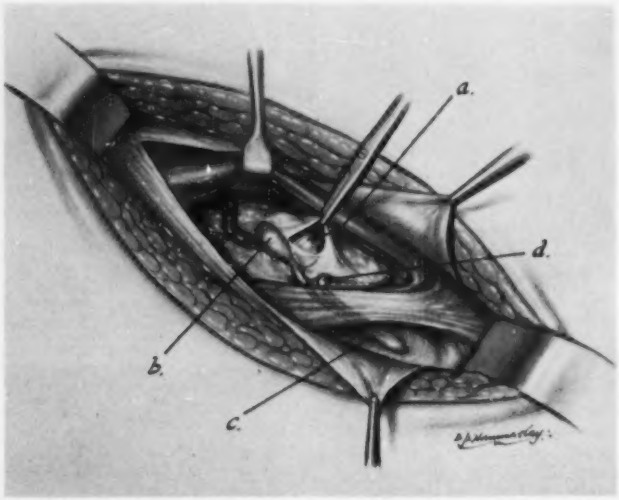


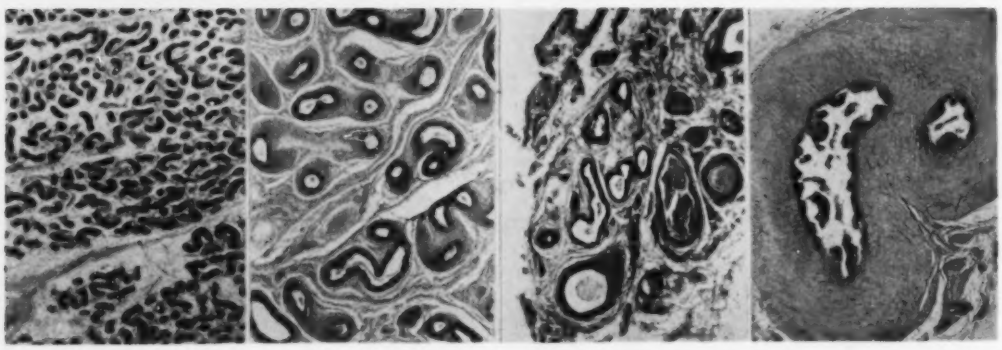
FIG. 713.—The histological appearances in *Case* I. Although there was no recognizable epididymis at operation, microscopy shows a tube system corresponding with the vasa efferentia and epididymis. A, Tubules lined with columnar epithelium about 1 in. away from the testis (× 90). B, A portion of testis showing a tube lined with columnar epithelium (v.e.), rete testis (r.t.), and seminiferous tubules (s.t.) (× 90).

vessels running to it, but its shorn-off appearance without an outwardly visible epididymis was quite distinctive and unique in this series. This testis would almost certainly have been overlooked without a very thorough inspection of the peritoneum from within along the line of testicular descent. Although this type of case is rare, its occurrence is not surprising, for it represents the theoretical converse to the cases of absent testis already described; the genital ridge has persisted but the mesonephric elements have developed imperfectly. No doubt both the gonad and the mesonephric duct can fail simultaneously, but this coincidence of two uncommon events must be exceedingly rare. Likewise it must be quite exceptional for there to be no evidence of one structure or the other to indicate to the surgeon the true state of affairs.

In our case of bilateral failure in development of the testes (*Case* 9), it seems probable that this was not a true bilateral agenesis because a total failure would almost certainly result in a Turner's syndrome and this was not present. Presumably the testes had undergone complete atrophy at a comparatively late stage in embryonic life after they had exerted their normal influence in the development of the male form (Jost, 1953). If this argument is accepted, the appearances in our unilateral cases of complete absence were sufficiently close to those found in the bilateral case to indicate that they, too, are unlikely to be due to true agenesis; the presence of well-developed spermatic vessels suggests that at some stage the testes were present and that complete atrophy had taken place, but until more evidence is available we prefer to avoid terms such as 'atrophy' or 'agenesis'.

In 8 of the cases of absent testis, the abnormality was strictly confined to one side and in the ninth an identical failure of development was present on each side, but in the patients with abdominal testis 4 cases out of 10 showed ectopic positioning of the testis on the opposite side. The cases are too few to draw any valid conclusions, but this does provide some evidence that the states of congenital absence and retention within the abdomen are unrelated and not merely degrees of the same state. In the cases of abdominal testis the high incidence of associated abnormality on the opposite side may indicate that some general influence has been at work; moreover it suggests that retention in the abdomen and inguinal ectopic positioning of a testis may be degrees of the same process. In the descent of the testis the gubernaculum initially guides the processus vaginalis to its final destination; the testis follows the





a *b* *c* *d*

FIG. 714.—A case of 'elongated epididymis' (*Case* E). The testis and epididymis are seen exposed in the iliac fossa. The epididymis extends through the internal ring and well into the inguinal canal and loops back upon itself to be continued as the vas deferens which follows a normal course to the pelvis. Representative histology is also shown. *a*, The testis (× 20). *b*, The epididymis (× 20). *c*, Continuation of epididymal tube system into the inguinal canal (× 20). *d*, Vas deferens (× 24·8).

same pathway, and if this goes to an abnormal site the testis will either follow it to an ectopic position or, if migration is incomplete, it may remain mobile within the abnormally placed processus to become an 'emergent' testis. Failure or abnormality of the gubernaculum, perhaps conditioned by inadequate endocrine activation in embryo, would appear the likely common factor. In cases of unilateral testicular absence, on the other hand, it is tempting to suggest that the testis fails to develop owing to infarction of the blood-supply even though spermatic vessels could be recognized in all cases but one. The well-developed, normally placed gonad on the other side makes any hormonal deficiency unlikely.

In 3 cases of abdominal testis a point of special interest was noted: the vas deferens could be traced from the pelvis into the inguinal canal, where it continued as a tubular structure which looped back into the abdomen to join the testes (*Cases* E, F, J, and *Fig.* 714). This formation has been described by Moschcowitz (1910), Mixter (1924), and, more recently, by Fowler and Stephens (1959), who, following the expression used by Mixter, termed it a 'long loop' vas. Histological study in 2 of our own patients in whom orchidectomy was performed shows that a tube system resembling the epididymis extended down to the apex of the loop (*Fig.* 714) and for this reason we suggest the term 'elongated epididymis'. (Incidentally, Fowler and Stephens suggest that orchidectomy can usually be avoided in this state. They present a study of the blood-supply to the testis and report their experience with mobilization of the testis by high division of the spermatic vessels.) It is important to remember that it is quite possible for the epididymis to take this form and be mistaken for an atrophic testis and be removed as such, leaving the testis within the abdomen.

Some years ago we encountered one such case when it presented as torsion of an abdominal testis; a few months previously an 'atrophic testis' had been removed from the inguinal canal, leaving behind the abdominal testis. The 'atrophic testis' was either one of a duplex testis (such as described by Wilson and Littler, 1953) or was in fact no more than an elongated epididymis; on the evidence available, the latter seems the more likely. We believe that the surgical assessment in any of these cases is not complete until the vas has been identified and traced, if necessary, through its continuation as an elongated epididymis to a point where the spermatic vessels can be seen joining it; this may entail exploration of the inguinal canal and the iliac fossa, but the presence of an apparent 'double vas' is distinctive and easily detected if looked for.

It is not easy to ascertain the frequency of occurrence of unilateral absence of the testis, but it is perhaps not so rare as is commonly supposed. During the period over which the cases presented were encountered, approximately 80 other patients, carefully selected, were operated on for ectopic or emergent undescended testis. Thus, out of 99 patients with abnormal testicular descent coming to surgery, 19 were either unilateral retention within the abdomen or total absence of the testis; 1 case in 5 showed one or other of these conditions, so that it is not a surgical problem of great rarity. In the present state of knowledge, it seems clear that the unilateral abdominal testis should be identified and brought to the scrotum before puberty or removed, and that it is wisest, in cases of unilateral absence of the testis, to remove the termination of the vas and associated tissue because the likelihood of malignancy in this site is unknown.

SUMMARY

There is good evidence that the abdominal testis is subject to an appreciable risk of malignancy and its removal from the abdomen is most desirable. The dilemma created by failure to locate a suspected abdominal testis is resolved if the vas deferens is used as a guide. This will lead the surgeon either to the testis or to a blind ending which provides positive evidence of a true absence of the testis on that side. The value of this method is demonstrated in 8 cases of unilateral absence of the testis; the presence of the vas deferens is a constant feature in this state.

Evidence is produced for regarding failure of development as a process distinct from abdominal retention of the testis. The latter would appear to be related to the various types of ectopic descent. Attention is drawn to the pitfall offered by an 'elongated epididymis' extending well below the abdominal testis into the inguinal canal.

Acknowledgements.—This investigation has been undertaken in collaboration with Professor S. D. M. Court and the Staff of the Department of Pædiatrics of the University of Durham.

I am grateful to Mr. C. J. L. Thurgar, Director of the Newcastle upon Tyne Regional Cancer Service, Mr. G. Dillingham, Secretary of the Registration Bureau, and to many surgical colleagues for their help in obtaining details of malignant disease of the testis in the north of England.

My thanks are due to Mr. D. P. Hammersley for the illustrations.

APPENDIX

It was found from the records of the Registration Bureau of the Newcastle upon Tyne Regional Cancer Service that, of 91 consecutive cases of malignant testes occurring over 5 years, 9 arose in abnormally descended testes; these all occurred after the age of 20 years. Therefore, during a period of 50 years, 90 malignant testes might be expected to arise from amongst the adult undescended testes in the north of England. Now, if the population of this region is approximately 3,000,000 and 0·2 per cent have undescended testes, then over any 50-year period there is a 'pool' of 6000 men with undescended testes at risk. During this time, 90 are likely to develop a malignant testis so that there is a 90 in 6000 (or 1 in 66) chance of any individual developing malignancy in his undescended testis during an adult life span of fifty years. During the same period, 820 out of 2,994,000 (or 1 in 3654) men might be expected to develop malignancy in normally descended testes.

No pretence is made that these figures are other than a gross approximation; they are intended to provide only a useful hint of the risks involved. The Registration Bureau believe that their coverage of malignant testes is 80 per cent complete for the area they serve. However, no allowance has been made in the calculations above for the possibility of some cases

not reaching the Bureau's records, and for this reason the risks of malignant change arrived at will err on the side of understatement.

Addendum.—Since submitting this paper 4 additional examples of unilateral absence of the testis have been encountered. In 2 of these the vas deferens ended blindly in the iliac fossa. In the other 2 cases the vas ended in the scrotum, and in both of these patients it was possible to palpate the vas in the neck of the scrotum prior to a confirmatory exploration.

Four further patients with abdominal testis have come to operation. Two of these patients had bilateral abdominal testes, but it proved possible to bring the testes to a scrotum position. Three further examples of 'elongated epididymis' were found.

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MEGALOBLASTIC ANÆMIA FOLLOWING OPERATIONS ON THE SMALL INTESTINE

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ANÆMIA, megaloblastic or macrocytic in character, has been described in association with strictures, anastomoses, or resection of the small intestine. Examples are reported by Thompson (1953), Pearson (1954), Thompson and Ungley (1955), and Evans (1956). Cameron, Watson, and Witts (1949a) also describe a case and review the earlier literature.

During the past eighteen months 3 cases of severe megaloblastic anæmia following operations on the small bowel have been encountered. These cases are described and the pathogenesis of the anæmias discussed in this paper.

CASE REPORTS

Case 1.—A 34-year-old woman developed a fæcal fistula following appendicectomy in 1935. This healed spontaneously in 1940. She remained well until 1948, when an abscess developed beneath the scar. This continued to discharge intermittently after incision. A barium meal on Oct. 12, 1950, showed marked narrowing and rigidity of the terminal ileum, which communicated with the skin. The appearances were suggestive of tuberculosis or Crohn's disease. At operation, on Nov. 11, the proximal ileum was distended and the terminal portion thickened and narrow. The proximal part was divided, the distal end closed, and an end-to-side ileotransverse colostomy performed. After this the fistula healed and she enjoyed good health.

In 1952 the fistula reappeared and was accompanied by diarrhœa. A barium meal revealed changes in the terminal ileum consistent with Crohn's disease, but the fistula was not outlined. The fistula was excised and the right half of the colon resected in November, 1952. The wound healed normally and her general health improved.

In March, 1956, she was admitted as an emergency with severe right-sided abdominal pain. At operation, distended coils of ileum, firmly bound by adhesions, were removed and a new anastomosis made. On discharge her hæmoglobin was 82 per cent (13·1 g. per cent) and she was in reasonable health.

She was admitted for the last time in October, 1957, complaining of diarrhœa and loss of weight and energy for several months.

Clinical examination showed wasting and pallor and an incisional hernia situated in the right iliac fossa.

Laboratory Investigations.—Hæmoglobin, 52 per cent (8·3 g. per cent); film, macrocytic; bone-marrow smear, megaloblastic erythropoiesis; total serum-protein, 4·7 g. per cent, albumin, 1·4 g. per cent, globulin, 3·3 g. per cent; serum-calcium, 7·5 mg. per cent; fæcal fat, 10 per cent (dry weight); test-meal, free acid present.

Radiological Investigations.—A barium meal and enema suggested the presence of a fistula between the jejunum and sigmoid colon.

PROGRESS.—A reticulocyte response of 20 per cent followed the administration of oral folic acid (60 mg. per day). Three pints of whole blood were given before operation on Dec. 6.

At operation, only one small adhesion was found between the small bowel and abdominal wall. About 7 ft. (213 cm.) of small intestine, mainly jejunum, remained and the anastomosis was satisfactory. Apart from a little perirectal thickening, the entire bowel was healthy and no fistula was found.

After operation the anorexia and wasting and diarrhœa persisted and she gradually deteriorated and died on

Jan. 3, 1958, from bronchopneumonia. Consent for autopsy was not granted.

Case 2.—A 26-year-old woman was operated on in November, 1951, because of attacks of pain in the right iliac fossa. Crohn's disease involving the terminal 3 ft. (91 cm.) of ileum was diagnosed. The ileum was divided proximal to the lesions and an ileotransverse colostomy performed.

After a few months the pain returned and was accompanied by diarrhœa. A barium meal in April, 1952, showed the anastomosis to be functioning well, but dilated loops of ileum were present. She improved on conservative treatment, but at this stage a chest radiograph revealed active pulmonary tuberculosis and she was transferred to a sanatorium. A year later the tuberculosis was under control and she had no abdominal symptoms.

After another year of good health she was admitted in May, 1954, with suspected intestinal obstruction. A barium meal showed the anastomosis to be situated in the jejunum and a loop of small bowel was outlined just below it, which retained the barium after 48 hours. Her symptoms disappeared with conservative treatment and she was discharged; the hæmoglobin was 70 per cent (10·4 g. per cent).

She was admitted to a medical unit in May, 1958, complaining of weakness and diarrhœa for several months.

Clinical examination showed marked anæmia, but there was no wasting or other evidence of malnutrition.

Laboratory Investigations.—Hæmoglobin, 34 per cent (5·0 g. per cent); film, macrocytic; bone-marrow smear, megaloblastic erythropoiesis; total serum-protein, 5·9 g. per cent, albumin, 3·6 g. per cent, globulin, 2·3 g. per cent; serum-calcium, 9·0 mg. per cent; serum inorganic phosphate, 3 mg. per cent; glucose-tolerance test, normal curve; fæcal fat, 24 per cent (dry weight); test-meal, histamine-fast achlorhydria.

Radiological Investigations.—A chest radiograph showed healed pulmonary tuberculosis. A barium meal and enema indicated that the anastomosis had been made at the jejuno-ileal junction. The bowel appeared healthy proximal to the anastomosis, but distal to it there was a blind pocket of small bowel about 6 in. (15 cm.) long.

PROGRESS.—She was given oral folic acid (60 mg. per day) and parenteral vitamin B_{12} (250 μg. per day). This produced a reticulocyte response of 22 per cent. On discharge, the hæmoglobin was 78 per cent (11·5 g. per cent) and she felt much better. Since then she has been receiving one injection of 250 μg. vitamin B_{12} every month. In August, 1959, her hæmoglobin was 92 per cent (13·6 g. per cent) and she was in good health apart from having one or two loose motions every day.

Case 3.—A 27-year-old man who, in 1950, had ligation of a patent ductus arteriosus, developed pulmonary tuberculosis in 1954. He developed intestinal obstruction and was admitted to the South Western Hospital, London. At operation on Sept. 1, 1954, multiple tuberculous strictures involving 16 ft. (486 cm.) of ileum were found. An ileotransverse colostomy was made without dividing the bowel. After operation his condition improved, but as colicky pain persisted the small bowel was divided distal to the anastomosis. This operation produced considerable relief.

He remained well until September, 1957, when he developed abdominal pain and distension and diarrhœa. Radiological examination revealed no evidence of obstruction or strictures and the anastomosis was satisfactory. He continued to have intermittent abdominal pain and diarrhœa, but was able to follow his employment.

On Jan. 2, 1959, he collapsed at work and was admitted, as an emergency, to St. Luke's Hospital. He gave a history of attacks of abdominal pain and vomiting and diarrhœa during the past month.

Clinical examination showed marked anæmia and slight icterus, but no evidence of malnutrition was present. There was abdominal distension and visible peristalsis. He settled down on conservative treatment and the anæmia was investigated.

Laboratory Investigations.—Hæmoglobin 46 per cent (6·8 g. per cent); film, macrocytic; bone-marrow smear, megaloblastic erythropoiesis; serum-bilirubin, 3·1 m$_{5}$. per cent; total serum-protein, 5·7 g. per cent, albumin, 3·8 g. per cent, globulin, 1·9 g. per cent; serum-calcium, 9 mg. per cent; serum inorganic phosphate, 3 mg. per cent; test-meal, free acid present; fæcal fat, 56 per cent (dry weight).

Radiological Investigations.—A barium meal, barium enema, and small-bowel barium enema were carried out and the following conclusions were reached: "The results are difficult to correlate, but there are at least two blind loops, one formed by cæcum, ascending colon, and proximal transverse colon, the other by a pouch of small bowel at the level of the anastomosis with the colon. It is possible that there is a third blind loop formed from small bowel below the point where it is anastomosed to the colon. The pouch mentioned at the anastomosis is probably sufficient on its own to account for the diarrhœa and anæmia."

PROGRESS.—Parenteral vitamin B_{12} (1000 μg. daily for 3 days, then 250 μg. weekly) produced a sub-optimal reticulocyte response and the hæmoglobin only reached 59 per cent (8·6 g. per cent) after three weeks' therapy. Vitamin B_{12} was replaced by oral folic acid (15 mg. daily) and this produced a reticulocytosis of 15 per cent. On discharge the hæmoglobin was 92 per cent (13·6 g. per cent) and he was feeling much better.

He was last seen in October, 1959, when the hæmoglobin was 92 per cent (13·6 g. per cent). Although he is able to follow his employment, he still has intermittent abdominal pain and diarrhœa. He is taking 10 mg. of folic acid daily.

DISCUSSION

The anatomical lesion in *Case* 1 is loss of ileum. Diminished absorption of hæmopoietic material due to lack of ileum is thought to be the cause of the anæmia. The loss of weight and the reduced values for serum-protein and calcium suggest that malabsorption was widespread. It is interesting to note that there was no evidence of steatorrhœa.

Isotope studies by Booth and Mollin (1959) show that vitamin B_{12} is absorbed from the ileum and that absorption is reduced and megaloblastic anæmia develops when the ileum is resected or short-circuited. This is also illustrated by a patient from the series of Thompson and Ungley (1955), who developed megaloblastic anæmia after resection of ileum. Massive oral doses of vitamin B_{12} failed to produce any improvement, but a good response was obtained by parenteral therapy.

In *Case* 1 there was no achlorhydria, thereby excluding Addison's anæmia. None of the other recognized causes of megaloblastic anæmia were present—namely, pregnancy, steatorrhœa, gross dietary deficiency, or anticonvulsant therapy. The onset of anæmia was eighteen months after the resection of ileum. These considerations suggest that loss of ileal absorbing surface was the cause of the anæmia.

In *Cases* 2 and 3 there is an anastomosis between the proximal small intestine and the transverse colon. In each patient a blind pouch or cul-de-sac of small bowel has developed at the site of the anastomosis.

Stagnation of the contents of a blind pouch or multiple diverticula favours the growth of an abnormal bacterial flora which tends to colonize the remaining small bowel. These organisms, by metabolic competition, defective synthesis, or toxin production, are

believed to deprive the host of hæmopoietic material resulting in the development of megaloblastic anæmia (Witts, 1951; Blatchford and Dawson, 1956; Gellman, 1956; and Scudamore, 1958). Sterilization of the bowel by the administration of broad-spectrum antibiotics causes remission of the anæmia (Evans, 1956; Scudamore, 1958). A similar anæmia has been produced in rats by the experimental formation of a cul-de-sac of small bowel (Cameron, Watson, and Witts, 1949b).

A histamine-fast achlorhydria is present in *Case* 2, but Addison's anæmia is unlikely to have developed at 26 years of age (Whitby and Britton, 1957). Free acid was present in *Case* 3. As the other causes of megaloblastic anæmia listed above were absent in these two cases, it may be assumed that the megaloblastic anæmia was caused by the intestinal blind pouch.

There was no clinical or biochemical evidence of widespread malabsorption in *Cases* 2 and 3. A random sample of fæces from *Case* 3 showed an increased fat content. Evidence of steatorrhœa was absent in *Case* 2. Cameron, Watson, and Witts (1949a), after reviewing previously reported cases, conclude that the presence of steatorrhœa is inconstant and it is not necessary for the development of megaloblastic anæmia. They note that steatorrhœa usually occurs in patients with a high anastomosis.

The observations of Booth and Mollin (1959) suggest that vitamin B_{12} is the treatment of choice in megaloblastic anæmia associated with resection of the ileum. A good reticulocyte response was obtained in *Case* 1 with oral folic acid, but her general condition deteriorated and she died from an intercurrent infection.

In *Case* 2 parenteral vitamin B_{12} and oral folic acid produced an adequate hæmatological response and she has been maintained on vitamin B_{12} alone for over a year. As free acid was absent, folic acid was not given alone because of the theoretical risk of precipitating subacute combined degeneration of the spinal cord. A sub-optimal response to parenteral vitamin B_{12} was obtained in *Case* 3, but subsequent oral folic acid produced a maximal reticulocytosis and he has been maintained on folic acid alone for nearly nine months.

A hæmatological response following the administration of parenteral liver extract, vitamin B_{12}, or oral folic acid has been reported in the literature (Cameron, Watson, and Witts, 1949a; Thompson, 1953; and Thompson and Ungley, 1955). The relationship between vitamin B_{12} and folic acid is complex and deficiency of either one or both may be present. The majority of the reported cases appear to have responded to vitamin B_{12}.

Surgical restoration of the normal anatomy of the intestinal tract produces improvement in the anæmia (Cameron, Watson, and Witts, 1949a). This was not attempted in *Cases* 2 and 3 as both patients responded well to medical treatment.

Routine prophylactic administration of vitamin B_{12} and folic acid is hardly practicable after operations on the small bowel, and prolonged treatment with antibiotics is undesirable. It is suggested that routine blood examinations, perhaps at half-yearly intervals, should be performed after small-bowel operations. This procedure would reveal the development of anæmia at an early stage.

SUMMARY

Three cases of megaloblastic anæmia developing after operations on the small intestine are reported.

In the first case, decreased absorption of hæmopoietic substances due to extensive resection of ileum was the cause of the anæmia.

The anæmia in the other two cases was associated with the development of blind pouches of small bowel at the site of an anastomosis.

The pathogenesis and treatment of the anæmia are discussed.

Acknowledgements.—We wish to thank Drs. Davidson and Rae and Mr. Whyte Watson for permission to publish these cases. The radiograph reports were furnished by Drs. Campbell, Carr, Franklyn, and Jay. We are grateful to the laboratory staff for technical assistance.

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DUPLICATION OF THE ŒSOPHAGUS*

REVIEW AND DESCRIPTION OF TWO CASES

By JOHN BORRIE

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GENERAL REMARKS

DUPLICATIONS of the alimentary tract are spherical or elongated hollow structures with three essentials:—

1. They are intimately attached to the alimentary tract;

2. They are lined by mucous membrane similar to some part of the alimentary tract; and

3. They possess a smooth muscle coat.

They may occur at any level from mouth to anus, but are most common in relation to the small intestine.

In 1953 Gross commented on 68 such lesions, of which 16 were thoracic. Thirteen arose from the œsophagus, while 3 were long tubular diverticula lying within the thorax but arising from the duodenum or jejunum.

While some such diverticula may be directed away from the alimentary canal, the majority are intimately bound in the muscle layer to the tract. Eighty per cent, however, do not communicate with the lumen.

The smooth muscle coat, having one to three layers, distinguishes the anomaly from chylous or lymphatic cysts. Though the epithelium always resembles some part of the alimentary canal, it is not always the same as that lining the subjacent gut. In the thorax œsophageal duplications are frequently lined by gastric mucosa.

Depending on the site and form of the duplication, so its size and shape vary from a globular swelling that declares its presence by pressure on vital organs as in *Case* 1, to a wandering tube up to 30 cm. long as in *Case* 2 reported here.

The Fluid.—This varies with the presence or absence of an alimentary communication. *If no communication exists*, it may be clear, colourless, and mucoid—sometimes cholesterol-containing; and where hæmorrhage has occurred, it may be brownish or blood-stained. *When a communication exists*, the fluid corresponds to that in the alimentary canal at that level, and usually allows of regurgitation of gas which gives a typical air shadow when investigated by radiographs.

Embryology.—Duplications are congenital in origin and several theories have been advanced for their formation. They can conceivably arise by *sequestration* of a group of cells from the developing gut.

Lewis and Thyng (1907) advanced the theory that they are the remains of embryological diverticula that normally regress but may become pinched off to give an adjacent duplication. Brewer (1944) feels that in the stage of vacuolation of the developing gut some of these vacuoles do not communicate with the definitive alimentary canal but remain as isolated units which form cysts or linear vacuoles that coalesce to form a parallel tube. This theory certainly explains the common musculature so often seen, besides the variety of epithelium.

CLINICAL FEATURES

Onset.—Most declare their presence in childhood. Gross (1953) found 65 per cent in the first year of life, an additional 9 per cent in the second year, and 8 per cent in the third year.

Symptoms.—In general these fall into four groups:—

1. *Obstruction* of the adjacent alimentary canal or other vital structures from lateral growth of the duplication;

2. *Pain* from fluid distension of the duplication;

3. *Local bleeding*, or necrosis from pressure on mesenteric blood-vessels;

4. *Slow perforation* from acid erosion of the duplication.

Duplications arising in the Thorax.—As fluid accumulates in the lumen these duplications steadily increase in size and cause symptoms by pressing on the respiratory tract. They can lie centrally in the mediastinum or more usually lie to one side, and have been reported twice as commonly and as large on the right as the left. They rarely communicate with the œsophagus. They are usually lined by gastric mucous membrane which secretes fluid containing renin, pepsin, and hydrochloric acid, often with a low *p*H value. Adherence to the lung can cause repeated hæmoptysis. Gross reported one which developed a fistula into the left upper lobe leading to lobar destruction.

Physical signs fall into three groups, namely, a space-occupying lesion, respiratory compression, or pleural fluid. One of the cases reported here had severe respiratory compression.

Duplications arising in the Abdomen but extending into the Thorax.—Arising from the duodenum or jejunum, they pass up behind the liver, through the right leaf of the diaphragm and into the posterior mediastinum.

Symptoms, which arise from distension of the track by fluid and gas, are cardio-respiratory in type and intermittent in nature. In addition, if the tube is lined by gastric mucosa, chronic ulceration may lead to anæmia, melæna, and sometimes severe hæmatemesis. The second case reported here was of this type.

INVESTIGATIONS

1. Radiography.—

Those arising in the Thorax.—Plain films reveal a space-occupying lesion in the posterior mediastinum. Barium studies show its relation to the œsophagus and whether or not there is a communicating opening. Bronchography establishes its relation to the lung.

* A paper read at the Thoracic Surgical Section of the General Scientific Meeting of the Royal Australasian College of Surgeons, Sydney, Aug. 21, 1959.

The occurrence of a fluid level may suggest a diaphragmatic hernia, sequestration of lung, or a hydatid cyst; but the possibility of a duplication should be kept in mind, as well as a possible origin below the diaphragm.

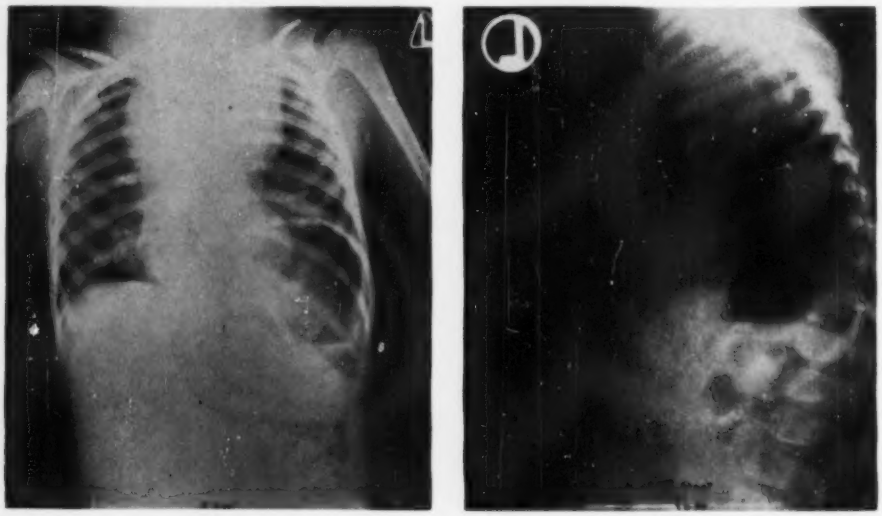
Those arising below the Diaphragm and extending into the Thorax.—Plain films reveal a mass usually in the lower mediastinum, and on the right side. With the patient standing up there is a fluid level which may closely simulate a hiatal diaphragmatic hernia, especially if barium refluxes into it. Œsophagoscopy should aid such a diagnosis by excluding any laxness of the cardia or reflux œsophagitis that often accompanies hiatal hernia.

2. Paracentesis.—Rarely for a posterior swelling needle aspiration can be both diagnostic and therapeutic, as in the first of these two cases.

TREATMENT

Because of their nature, growth, and complications, surgical excision provides the only satisfactory treatment.

Duplications arising within the Thorax.—These lesions are exposed by the most direct route, the pleura incised and reflected, the normal œsophagus above and below isolated and raised in a sling, and the lesion dissected. It is followed down to its neck and divided, often leaving a small portion of wall and mucous membrane attached. The mucous membrane is excised and the remaining smooth muscle sutured to reinforce the œsophageal wall at that point.



A B

FIG. 715.—Admission postero-anterior (A) and lateral (B) chest films showing a mass in the superior mediastinum compressing the trachea forwards. (*By courtesy of Messrs. H. K. Lewis.*)

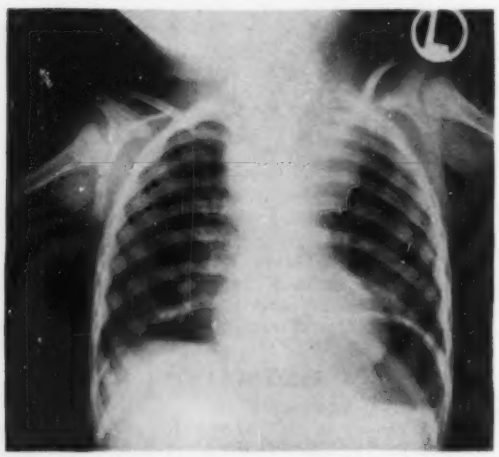


FIG. 716.—Following aspiration of 120 ml. through the third right intercostal space posteriorly, symptoms of tracheal compression were dramatically relieved. The mass became half size. (*By courtesy of Messrs. H. K. Lewis.*)

Duplications arising in the Abdomen but extending up into the Thorax.—The extent of the surgical procedure depends on: (*a*) the nature of the mucosa in the lesion; and (*b*) its size and ramifications.

Where the mucosa simulates small or large intestine, and the symptoms are due to a space-occupying lesion, then thoracic excision suffices. Where, however, it is gastric in nature and has caused severe bleeding, then total excision is required.

Regarding the surgical approach, if the lesion is in the lower mediastinum then a thoraco-abdominal approach can be used, remembering that the duplication usually passes behind the liver. In the abdomen it may be necessary to excise the junction region and perform end-to-end anastomosis as reported by Gross (1953). Where the lesion extends up beyond the

root of the neck, an additional higher intercostal incision may be necessary, as later described.

The purpose of this paper is to emphasize many of these points of diagnosis and management in the following two case reports, each of which presented unusual features of diagnosis and management.

CASE REPORTS

Case 1.—**Duplication arising in the Thorax.**

J. A. S. (North Region Chest Surgery Centre, Newcastle, England, Case No. 6473), a boy aged 3½ years, was seen in consultation in another hospital on June 9, 1950. There was a history of wheeziness since birth, a cough when teething, besides pneumonia when 1 year old, and again at 2½ and 3 years of age.

Recent History.—When kicking a football two weeks previously, he had been crushed by a playmate falling on top of him. He immediately complained of constant pain in the chest, and was wheezy, dyspnœic, and restless. He lost his voice. His cervical veins were swollen.

On Admission.—He was admitted to hospital and treated for tracheo-bronchitis. Chest radiographs showed a left phrenic nerve palsy, and a large mass in the superior mediastinum, which compressed the trachea to a slit and displaced both trachea and œsophagus forward and to the right (*Fig.* 715).

Because of the size and site of the tumour, the superior vena caval compression, and paralysed diaphragm, a diagnosis of mediastinal malignant tumour was made and an opinion sought as to the possibility of deep X-ray therapy. On June 9 he was transferred to the North Region Chest Surgery Centre for further treatment.

Further clinical examination revealed no lymphadenopathy. The blood-picture was normal, and there was no radiographic evidence of vertebral body erosion. Although at first malignancy seemed the inevitable diagnosis, yet because of the length of the history and his excellent state of bodily nutrition, this diagnosis was doubted. Because of the posterior position of the growth, the imminence of total tracheal occlusion, and because it was felt that some effort should be made either to get a biopsy and establish 'malignancy', or else to prove that the swelling was in fact cystic, it was finally decided to needle the swelling. There was nothing to lose and everything to gain. An aspirating needle was therefore inserted into the third right intercostal space posteriorly just beyond the vertebral transverse processes. Quite unexpectedly 120 ml. of cloudy straw-coloured fluid, which later became brownish in colour, were removed. The child immediately improved, breathed more evenly, and the signs of tracheal and superior vena caval compression dramatically disappeared. Post-aspiration chest films showed that the shadow was now half its original size (*Fig.* 716). A diagnosis of cyst (? œsophageal ? dermoid) was therefore made and early excision planned.

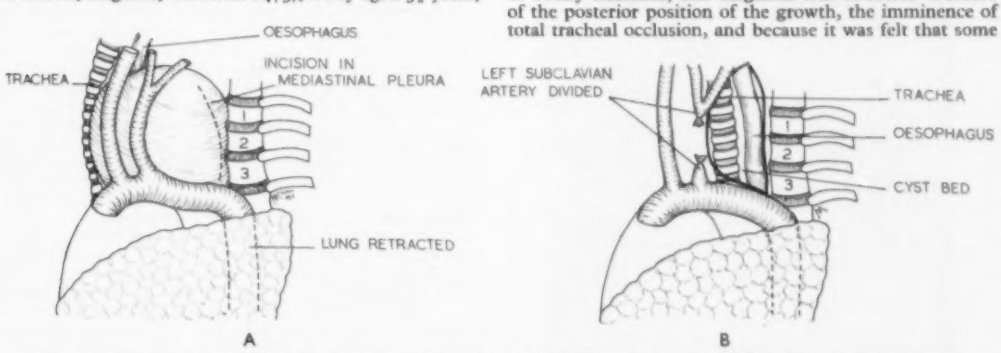


Fig. 717.—A, Drawing of the superior mediastinum from the left side, showing the cystic duplication displacing and compressing the trachea and œsophagus forwards. B, The left subclavian artery has been divided and the duplication excised.

Fig. 718.—The duplication after excision. Note its thick wall. (*By courtesy of Messrs. H. K. Lewis.*)

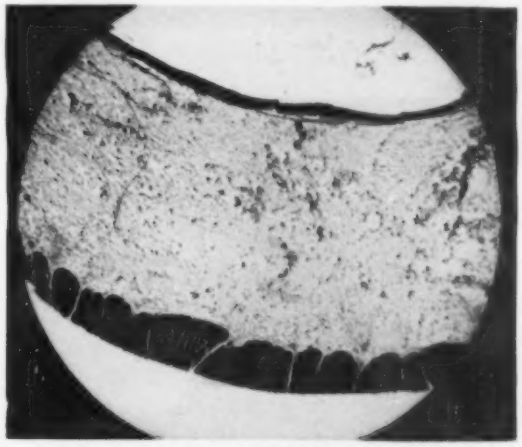


Fig. 719.—Photomicrograph of the three layers of the cyst wall. (*By courtesy of Messrs. H. K. Lewis.*)

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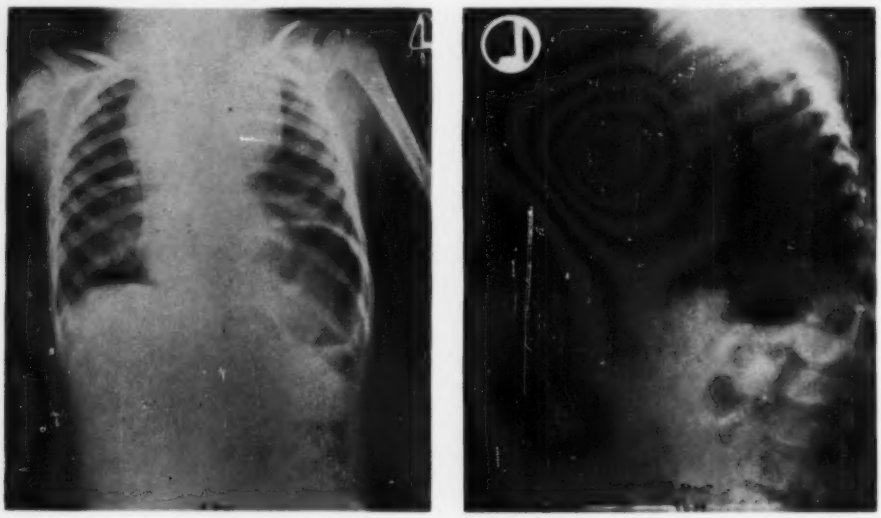

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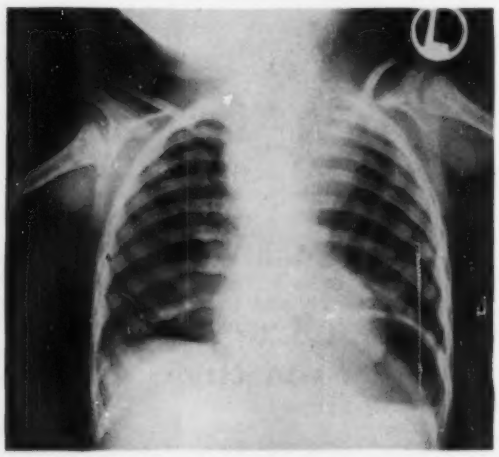

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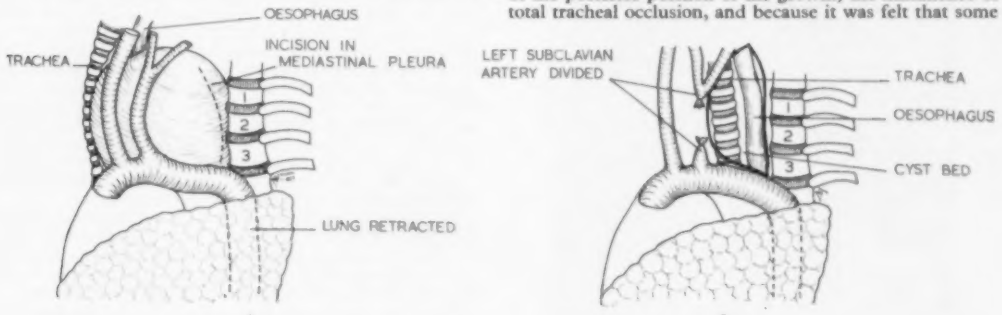

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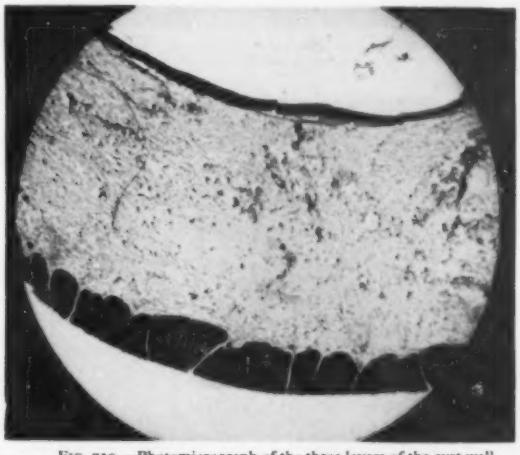

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Because of the size and site of the tumour, the superior vena caval compression, and paralysed diaphragm, a diagnosis of mediastinal malignant tumour was made and an opinion sought as to the possibility of deep X-ray therapy. On June 9 he was transferred to the North Region Chest Surgery Centre for further treatment.

Further clinical examination revealed no lymphadenopathy. The blood-picture was normal, and there was no radiographic evidence of vertebral body erosion. Although at first malignancy seemed the inevitable diagnosis, yet because of the length of the history and his excellent state of bodily nutrition, this diagnosis was doubted. Because of the posterior position of the growth, the imminence of total tracheal occlusion, and because it was felt that some effort should be made either to get a biopsy and establish 'malignancy', or else to prove that the swelling was in fact cystic, it was finally decided to needle the swelling. There was nothing to lose and everything to gain. An aspirating needle was therefore inserted into the third right intercostal space posteriorly just beyond the vertebral transverse processes. Quite unexpectedly 120 ml. of cloudy straw-coloured fluid, which later became brownish in colour, were removed. The child immediately improved, breathed more evenly, and the signs of tracheal and superior vena caval compression dramatically disappeared. Post-aspiration chest films showed that the shadow was now half its original size (*Fig.* 716). A diagnosis of cyst (? œsophageal ? dermoid) was therefore made and early excision planned.

AT OPERATION.—After pre-operative preparation with penicillin, 100,000 units twice daily, for his respiratory infection, left thoracotomy was performed on June 12 through the fourth left intercostal space. A large cystic swelling, 5 cm. in diameter, was found lying in the posterior mediastinum above the aortic arch, behind and to the left of the œsophagus, and in front of the vertebral column. After dividing the left subclavian artery, and further aspirating 20 ml. of thick mucoid fluid, the tumour was removed (*Fig.* 717) (surgeon, Mr. S. A. Mason).

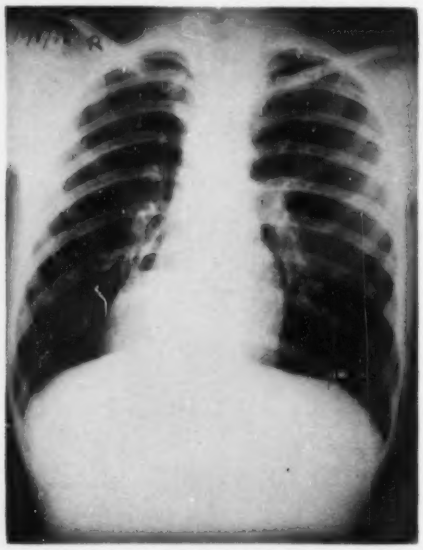
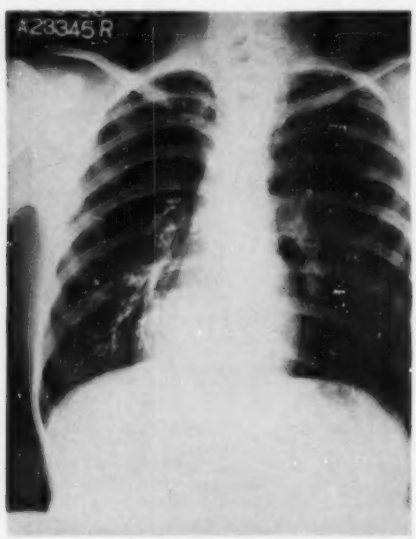


FIG. 720.—Radiograph showing air-containing cyst projecting beyond the right border of the heart.



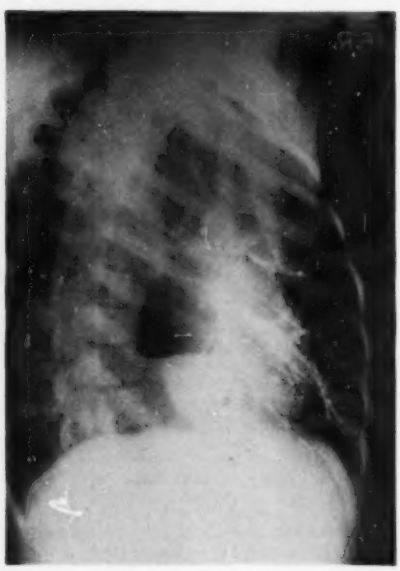


FIG. 721.—Bronchograms confirming that the cyst is extrapulmonary and lies in the posterior mediastinum.

The child made an uneventful recovery, and when reviewed 6 months later was in excellent health. The left phrenic nerve palsy had completely recovered.

PATHOLOGY.—Macroscopically the specimen consisted of a thick-walled cystic swelling 7 × 6 × 5 cm. in diameter. The wall was ½ cm. in thickness (*Fig.* 718).

Microscopically the cyst wall had the characteristic structure of gut wall, i.e., stratified squamous epithelium, muscularis mucosæ, submucous connective tissue, nerve plexus, and circular and longitudinal muscle layers, closely resembling œsophagus in its lower third (*Fig.* 719).

Comment.—This case follows the more usual pattern emphasized by Gross, and appeared within the first three years of life. Serious symptoms arose as the duplication grew within the unyielding thoracic inlet, to cause severe tracheal and jugular venous compression. Although this compression, together with the mediastinal shadow and paralysed left diaphragm, all apparently supported the diagnosis of 'mediastinal lymphosarcoma', nevertheless careful analysis of his history of repeated respiratory episodes over the preceding three years made this supposition less likely. Final diagnosis came from aspirating fluid through the third right intercostal space posteriorly, relieving pressure and obstructive symptoms.

The surgical approach was facilitated by dividing the left subclavian artery in its first part. Finally, the fallacy of believing that paralysis of the diaphragm occurs *only* with malignant neoplasms is refuted by this case, where the nerve functioned again 6 months after operation.

Case 2.—**Duplication arising in the Abdomen but extending to the Thorax.**

H. D., a sheep farmer's son, aged 12 years, was admitted to Wakari General Hospital, Dunedin, on Oct. 6, 1958, with a long history of recurrent bronchitis. For years he had suffered from a winter cough accompanied by yellowish, thick sputum. He was frequently absent from school.

Three years previously he noticed a midline retrosternal chest pain—burning in type and often relieved by drinking warm water. It bore no relation to meals or activity, but did occur when lying down, especially in bed

at night. It was intermittent, never severe, and never required analgesia. Recently, after an attack of bronchitis, a mass miniature chest film showed an unusual right posterior mediastinal shadow (*Fig.* 720). This had grown since a normal film of June 17, 1955.

In addition, since 1955, a chronic microcytic anæmia was treated with fergon tablets, gr. 5 thrice daily.

PAST HISTORY.—His past history and family history were non-contributory.

PHYSICAL EXAMINATION.—The boy was thin and slight for his age. In his chest was an area dull to percussion below the angle of the right scapula but ventilatory function appeared normal. The cardiovascular, alimentary, genito-urinary, and central nervous systems were normal.

FURTHER INVESTIGATIONS.—

Chest Films.—These revealed a large cyst containing air and fluid, 11 × 8 cm., in the right lower hemithorax, extending across the midline in the posterior mediastinum to displace the œsophagus anteriorly and to the left as confirmed by barium studies. No lesion was detected in the stomach and duodenum. Bronchography confirmed that the cyst lay beyond the lung (*Fig.* 721). A barium enema was not performed.

Blood Examination.—This showed an eosinophilia of 10 per cent, a packed cell volume of 42, hæmoglobin of 13·5 g. per cent, and white blood-cell count of 7100 per cm. The hydatid complement-fixation test was negative.

The Casoni skin reaction was strongly positive.

DIAGNOSIS.—Because of the history of asthma and bronchitis all his life, his chronic iron-deficiency anæmia requiring constant iron, chest pain for three years, and radiographic evidence of a cystic swelling with a fluid level in the lower right chest posteriorly, thoracotomy was advised. The first medical and radiological opinion was 'hydatid cyst', the surgical opinion 'herniation of bowel'.

AT OPERATION (Oct. 9, 1958).—A right 6th-rib thoracotomy was performed and revealed a long cystic swelling, 8 × 5 × 5 cm., in the posterior mediastinum. This was dissected and at first thought to contain colon. Closer inspection showed it was a cystic dilatation of the œsophagus, which narrowed to its normal 2 cm. diameter at the level of the diaphragm and again at the level of the azygos arch. To help solve the riddle a wide-bore stomach tube was inserted through the mouth, but could not be felt passing down this dilated 'œsophagus'. It was found to lie in a normal second œsophagus lying parallel to the left of the lesion and quite separate from it. On squeezing the dilated right-sided 'œsophagus', mucus material disappeared below the diaphragm and regurgitated up the stomach tube (*Fig.* 722).

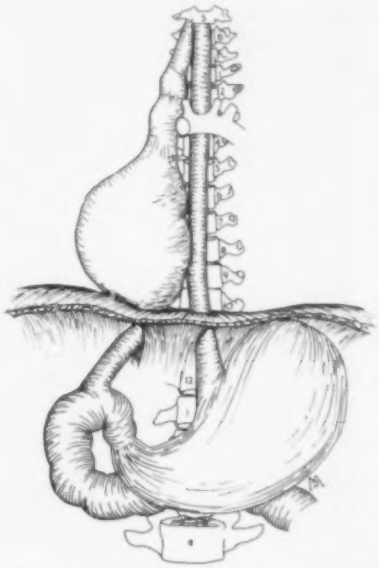
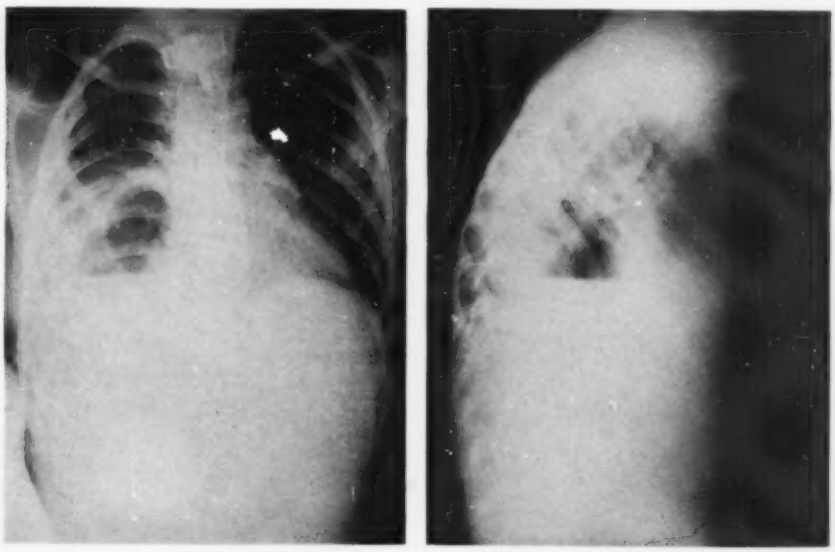


FIG. 722.—Composite drawing of the lesion, showing the intrathoracic portion seen at operation and the intra-abdominal portion which was disclosed by subsequent pleurograms during the convalescence (*see Fig.* 724).



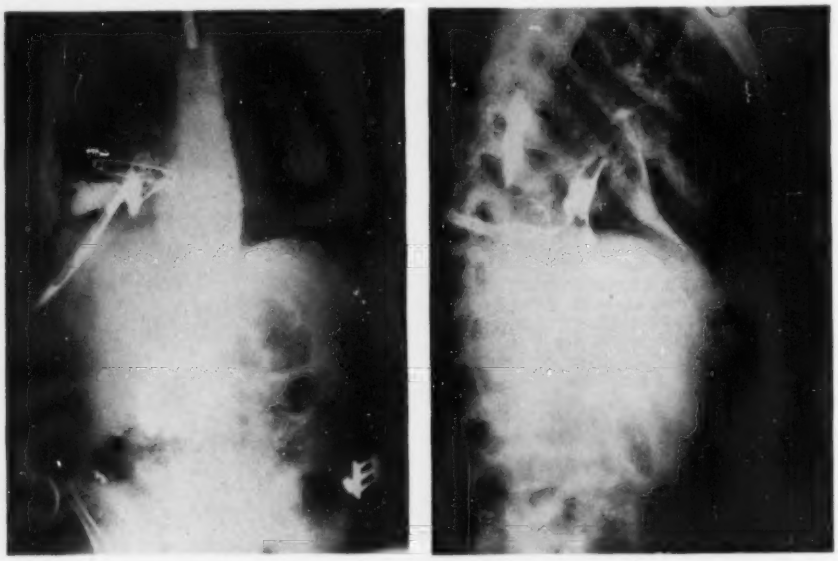
A B

FIG. 723.—Anteroposterior (A) and right lateral (B) chest films 9 days after operation, showing that a fluid-containing air space had now formed from rupture of the distal œsophageal stump.

A second intercostal incision was then made higher up through the 4th intercostal space, and the duplication traced proximally. On dividing the azygos arch it was found to extend right up to the level of the cricoid cartilage, where it became an attenuated strand adherent to the normal œsophagus posteriorly. It was divided here and found to have a lumen which, however, did not enter the normal œsophagus. The cervical stump was closed with interrupted No. 2 silk sutures.

Returning to the lower end, this was lifted up, clamped at diaphragmatic level, divided, and closed in two layers with interrupted silk sutures. The significance of this lower stump was at that time not fully appreciated.

Standard thoracotomy closure was performed.



A B

FIG. 724.—Anteroposterior (A) and right lateral (B) pleurograms 21 days after operation, confirming that the lower stump of the duplicated œsophagus was in free communication with the first part of the duodenum.

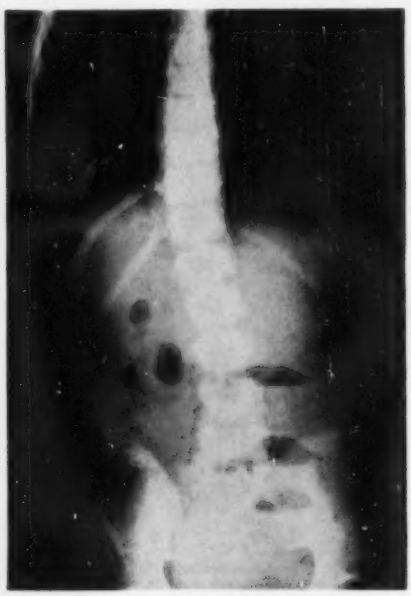


FIG. 725.—Abdominal radiograph showing fluid levels from small-bowel obstruction.

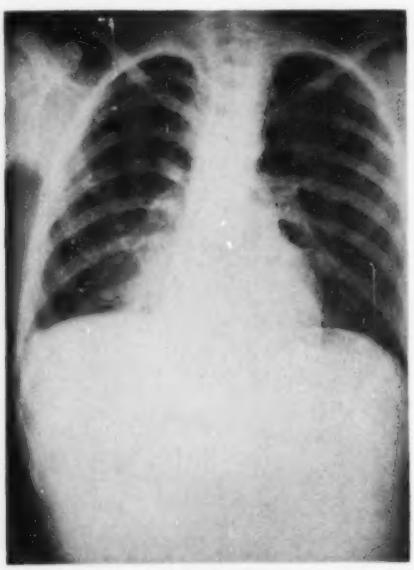


FIG. 726.—Chest radiograph (Dec. 30, 1958) taken when patient was finally discharged home.

POST-OPERATIVE COURSE.—This was unduly complicated. On the *first* post-operative day his right lung was fully expanded, but he would not cough. On the *second* day, because of sputum retention unrelieved by physiotherapy and bronchoscopy, tracheostomy was performed. His right lung fully re-expanded.

From the *fifth* to the *eighth* day he became febrile, and chest films now showed air at the base of the right pleural cavity (*Fig.* 723). This was relieved by an intercostal drain. The fluid proved to be succus entericus, and in the first hour 3600 ml. drained away. Because of this major and an intraperitoneal search made for the duplication stump, which was not detected either at the cardiac end of the stomach or along its lesser curvature.

The intercostal and rectal fluid loss proved copious. Jejunostomy feeding immediately caused intractable diarrhœa of at least six watery motions per day. Further, when fluid was given by mouth, it greatly increased the flow of succus entericus from the intercostal drain, up to 700 ml. per day. For example, on the sixteenth day intercostal drainage was 300 ml. and per rectum 1280 ml. On the twentieth day intercostal drainage was 1270 ml. and 640 ml. per rectum.

The following day a pleurogram showed that the stump of duplicated œsophagus led behind the liver to the junction of first and second parts of the duodenum (*Fig.* 724).

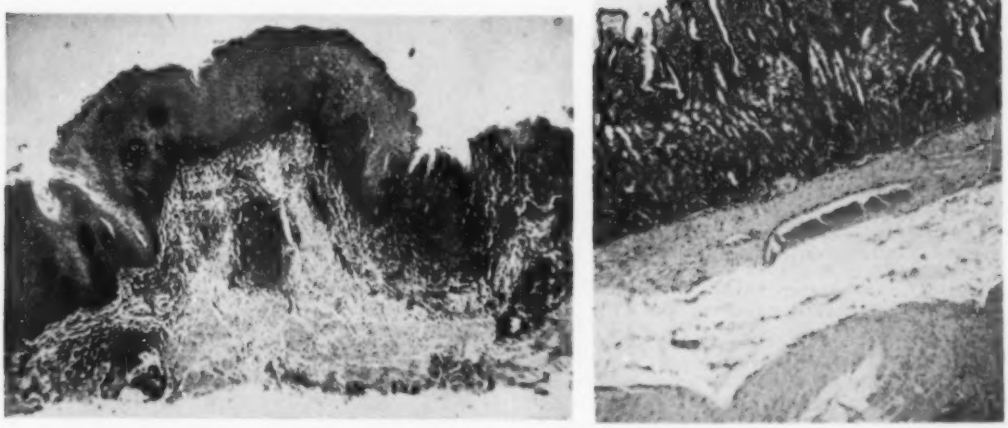
Convalescence was further complicated by the developing of a *bronchopleural fistula* on the thirteenth post-operative day, and this remained open and discharging succus entericus into the bronchi until the thirty-first post-operative day. Continual tracheostomy suction was therefore life-saving.

The tracheostomy tube was removed 37 days after insertion, and the intercostal tube was finally removed 42 days post-operatively. He was discharged home at the end of 60 days.

ON READMISSION.—Three days later, on Dec. 13, he was readmitted as an emergency with a history of mild colicky subumbilical pain which he had concealed for a week. The day before readmission he had eaten four green apples, immediately developed severe colic, and had commenced to vomit. The vomitus, at first blood-stained, and containing undigested apples, later became dark brown in colour.

On readmission he had the signs of subacute intestinal obstruction. Bowel-sounds were audible. He was considered to have a partial small-bowel obstruction which

FIG. 727.—Macroscopical view of the lesion, showing the large lower cyst lined by gastric mucous membrane and the proximal cyst lined by stratified squamous epithelium.



A B

FIG. 728.—A, The wall of the upper cyst lined by stratified squamous epithelium. (× 18.) B, The wall of the lower cyst lined by gastric type of mucous membrane. (× 18.)

fistulous leak from the intestinal tract, intravenous therapy was commenced and electrolytes were checked. The lower stump of the duplication, sutured at diaphragmatic level, had clearly broken down to give this enteropleural-cutaneous fistula. A jejunostomy was therefore performed,

was confirmed by a radiograph (*Fig.* 725). Because of his previous complicated illness a short period of conservative treatment was warranted, with a gastric tube, intravenous drip, and half-hourly observations. There was no result from an enema, and rectally no lesion was detected.

By Dec. 15, after 48 hours of observation, he still vomited intermittently, and abdominal pain was still present. As abdominal radiographs showed no alteration in the degree of intestinal obstruction, a laparotomy was advised and performed by my colleague, Mr. Victor Pearse.

LAPAROTOMY.—A lower paramedian laparotomy revealed some 500 ml. of serous fluid in the abdominal cavity. The jejunum was distended. A band obstruction was found stretching between two coils of jejunum, with a large coil of ileum herniating through this. The band was divided, the areas where it was attached were sutured and invaginated. The jejunostomy stoma was also closed. Closer inspection confirmed that he also had malrotation of the gut, with the duodenojejunal flexure to the right of the vertebral column just medial to the high cæcum.

POST-OPERATIVE PROGRESS.—During his second convalescence sputum retention, unrelieved by bronchoscopy, required the re-opening of his tracheostomy.

He was finally discharged 14 days after laparotomy. When seen 12 months later he was well, thriving, never fitter, and with a normal blood-count (*Fig.* 726).

PATHOLOGICAL REPORT.—Macroscopically the specimen consisted of a pear-shaped structure 13 cm. long, at its widest 7 cm. and at its narrowest 1·5 cm. in diameter. Vertical section revealed two cystic dilatations in the line of a muscular tube resembling œsophagus (*Fig.* 727).

The smaller proximal cyst measured 4 × 1·5 cm. It had irregular walls. The lower and larger cyst measured 8 × 6·5 × 4 cm. Its lining wall was rugose, resembling gastric mucosa, was 0·5 cm. thick, but had scattered areas of considerable thinning. These two cysts did not communicate with each other, but the lower and larger cyst communicated to the duodenum via the muscular tube into which it opened at its base.

HISTOLOGY (Dr. Derek Perry) (*Fig.* 728).—The upper extremity of the lesion was lined on its inner surface by thick squamous epithelium. The corium contained a dense infiltration with chronic inflammatory cells, which were also present in small aggregations in the smooth muscle layer outside. Most peripherally was a layer of dense collagenous fibrous tissue in which were many dilated blood-vessels. The central dilated part and the distal extremity were lined internally by mucosa of gastric type. Chronic inflammatory cells were present in the corium, muscle, and the outer layers, but not to the same extent as seen in the proximal extremity.

Conclusion.—A duplication of the œsophagus with proximal cysts lined by squamous epithelium and a larger distal one lined by gastric epithelium.

Comment.—Had the possibility of such a lesion been in mind, the correct diagnosis could have been deduced pre-operatively, especially when an air-containing lesion was found by bronchography to be extrapulmonary and by barium studies not to be a diaphragmatic hernia.

A barium swallow and meal showed no obvious connexion with the œsophagus and stomach, while a barium enema would have excluded the colon.

This particular lesion was dissected at operation to the origin of the œsophagus, but, although it had a subjacent cavity lined by squamous epithelium, there was no cervical communication with the alimentary canal. This communication, as shown later by a pleurogram, occurred below the diaphragm, at the junction of the first and second parts of the duodenum.

Gross (1953) believes that where such a form of duplicated œsophagus is discovered at operation, it should be traced to its junction with the normal bowel and divided there. This of course necessitates an abdominal extension of the incision. The complication encountered of a duodenopleuro-cutaneous fistula, with all the complicated control required for his disordered fluid balance, shows how carefully one must close the bowel stump, be it intra-pleural or intra-abdominal. The further complication of bronchopleural digestion by the duodenal fluid to form a bronchopleural fistula demanded the continued use of a tracheostomy until the duodenal fistula had closed.

At the time of the jejunostomy a search along the lesser gastric curvature for the duplicated œsophagus was unsuccessful. It lay farther to the right, and entered the duodenum.

The final post-operative complication, of band obstruction of the small intestine, allowed one to detect in addition a malrotation of the bowel.

In considering the advisability of removing the duplication the difference in this patient's physical condition one year after excision, his freedom from anæmia, and his progressive growth, are sufficient reasons for advocating such a course.

SUMMARY

The pathological and clinical features of duplication of the œsophagus are reviewed, together with methods of investigation and treatment. The contrasting features of duplications arising within the thorax and those arising in the abdomen but extending up to the thorax are stressed, and the management of a case illustrating each type fully described. The former, complicated by severe tracheal compression before operation, was temporarily relieved by aspiration of the cyst. The latter, after excision, was complicated by a duodenopleuro-broncho-cutaneous fistula and band obstruction in a malrotated gut. Both patients recovered.

Acknowledgements.—I am happy to record my obligations to Mr. George Mason, North Region Chest Surgery Centre, Newcastle upon Tyne, England, for permission to publish *Case* 1; to Mr. Victor Pearse, Dunedin, New Zealand, and Dr. Jack Chin for their great assistance with *Case* 2; to Mr. Gerald Brook of the Department of Photography, University of Otago, who kindly prepared the photographs; and to Miss Noeleen Richardson for all her willing secretarial assistance. I am also grateful to Miss Dorothyann McHugh for the line drawings.

I also thank Messrs. H. K. Lewis & Co., London, for the use of *Figs.* 715, 716, 718, and 719 from my article in Maingot's *Management of Abdominal Operations*.

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CARDIOSPASM

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The disease called 'cardiospasm' is a diffuse abnormality of œsophageal motility. The cause is not known and it is not known whether the pathological changes in the established case are primary or secondary. The majority of patients with cardiospasm die before their expected age of survival (Ellis, 1960) and as a direct consequence of one of the complications of the disease. Since the cause of cardiospasm is not known all the more successful forms of treatment are directed only at relief of distal œsophageal obstruction and in many instances it is only when distal obstruction has been relieved that the gross disorders of œsophageal peristalsis are revealed. The forms of treatment most frequently successful are cardiomyotomy (Heller's operation) and hydrostatic dilatation of the cardia. One or other of these procedures will, in 60–70 per cent of patients, sufficiently relieve distal œsophageal obstruction to make these patients believe that they are swallowing normally, to improve nutrition, and to prevent the development of the complications of untreated cardiospasm.

During the ten-year period, 1948–1958, 55 patients investigated in the Thoracic Surgical Unit in Edinburgh were found to have cardiospasm. The principal purpose of this paper is to assess the results of treatment in these patients, with particular reference to the changes that Heller's operation and hydrostatic dilatation of the cardia may produce on the radioscopic features of the disease. The technique of cardiomyotomy used in this Unit is described and some of the clinical features elicited in these patients are discussed.

Of the 55 patients investigated and treated 33 were females and 22 were males. In *Fig.* 729 there is shown the age range in the two sexes in relation to the type of treatment undertaken—dilatation of the cardia with the Negus hydrostatic dilator or Heller's operation. Heller's operation was undertaken in 26 patients, 15 of them female. From the graph it would appear that a disproportionately large number of young females were submitted to cardiomyotomy. The onset of symptoms in cardiospasm is, however, common in adolescents, the incidence of cardiospasm is slightly higher amongst females, and these young patients presented for a surgical opinion during the decade after Heller's operation had become popularly established as a successful procedure.

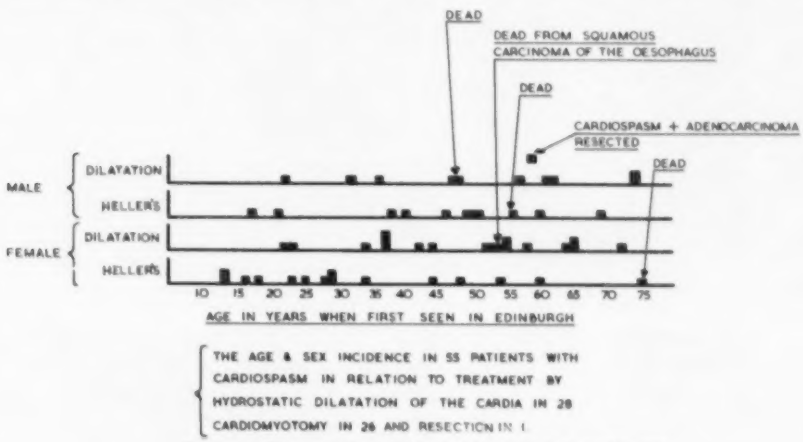

DEAD
DEAD FROM SQUAMOUS CARCINOMA OF THE OESOPHAGUS
DEAD
CARDIOSPASM + ADENOCARCINOMA RESECTED
DEAD
MALE
DILATATION
HELLER'S
FEMALE
DILATATION
HELLER'S
10 15 20 25 30 35 40 45 50 55 60 65 70 75
AGE IN YEARS WHEN FIRST SEEN IN EDINBURGH
THE AGE & SEX INCIDENCE IN 55 PATIENTS WITH CARDIOSPASM IN RELATION TO TREATMENT BY HYDROSTATIC DILATATION OF THE CARDIA IN 28 CARDIOMYOTOMY IN 26 AND RESECTION IN 1

FIG. 729.—The age and sex incidence in patients with cardiospasm.

The natural history of untreated cardiospasm is conveniently divisible into three phases (Ellis, 1960): a relatively short early stage of obtrusive symptoms; a long and relatively silent stage during which the patient learns to live with the disease; and the late phase of complications. The adolescent girls referred to above presented during the first stage of the disease and failed to show improvement with dilatation. Repeated œsophagoscopy in young people with a full dentition is never undertaken with enthusiasm, and Heller's operation was resorted to in these patients after a relatively short trial of dilatation. The elderly patients in this series began to have symptoms when they were adolescent, thirty or forty years ago, when treatment was usually conservative and consisted of little more than observation; they had become accustomed to their symptoms and presented, during the decade under discussion, late in the 'silent' phase of the disease or during the stage of complications.

In 28 patients the only form of treatment undertaken was dilatation of the cardia; 18 of these were female. One male was submitted to resection because the classical radiographic features of gross cardiospasm were complicated by histologically demonstrated and

surgically established adenocarcinomatous changes at the cardia.

At the outset, in 1948, the intention was to assess in patients with cardiospasm the results of hydrostatic dilatation of the cardia, and to undertake cardiomyotomy only when an adequate trial of dilatation had unusually large œsophageal diverticula (*Fig.* 730) the resection of which was considered important for the correction of œsophageal stasis. This patient had, in addition, severe rheumatoid arthritis. Heller's operation was therefore undertaken in 11 patients after an adequate trial of hydrostatic dilatation of

FIG. 730.—Cardiospasm in a man aged 69 years complicated by large œsophageal diverticula, at least one of which, as shown on the film on the right, retained barium with the patient in the erect posture.

FIG. 731.—The film on the right shows diminution in œsophageal calibre several years after relief of dysphagia by hydrostatic dilatation of the cardia in a patient with cardiospasm. The radioscopic appearances before treatment are recorded on the films on the left. The radiograph on the right was made immediately after the patient had drunk 6 fl. oz. of barium.

shown that this was insufficient for the relief of symptoms in the individual. This plan was frustrated in 15 patients. In 10 the cardia could not be found at œsophagoscopy; in 2 a biopsy from the cardia was interpreted histologically as showing the changes of squamous carcinoma, an interpretation shown subsequently to be erroneous; in 2 patients a transabdominal Heller's operation had been undertaken elsewhere without relief of symptoms, and these patients were referred for the specific purpose of submitting them to a more satisfactory transthoracic Heller's operation; in 1 patient in addition to cardiospasm there were the cardia had failed to relieve symptoms and in 15 patients without a preliminary trial of dilatation.

THE RESULTS OF HYDROSTATIC DILATATION OF THE CARDIA

In 28 patients the only treatment undertaken was hydrostatic dilatation of the cardia. Fourteen of these patients claim to be well and without symptoms at the present time, from two to twelve years after the last dilatation. Dilatation was undertaken five times over two years in 1 patient, four times over eighteen months in 3 patients, three times over eighteen

months in a further 3 patients, twice during a year in 2 patients, and in 4 patients only one dilatation was required for the relief of symptoms. Because of the return of symptoms three months before the second dilatation, the last of the 14 patients required a second dilatation five years after the original procedure.

The confidence with which these patients claim to be well and symptom-free can, in some, be shaken by persuasive questioning. All have gained weight and all eat relatively normal and varied diet, but most can be induced to admit that they eat more slowly than others in the family, some still indulge in trick manœuvres to hasten the passage of food, many avoid eating in public places because they find that their symptoms return in these circumstances, and some avoid certain foods such as the more fibrous types of fish because they find that such foods induce dysphagia.

Radioscopy in these 14 patients shows an appreciable improvement in comparison with pre-operative appearances (*Fig.* 731). There is in all, as after Heller's operation, intermittent and sometimes gross œsophageal spasm, and peristalsis is inco-ordinated, but barium passes rapidly through the cardia and the œsophagus is empty within three minutes of taking 6 fl. oz. of barium. The calibre of the œsophagus is normal or only slightly dilated.

Two patients treated only by dilatation have died. A man, 48 years old when he was first seen in 1952, was relieved of dysphagia by a single dilatation. He died in 1960 from coronary thrombosis, having had angina of effort for several years before this. At autopsy the opportunity was not used for examining the œsophagus in detail. A woman of 54 had dysphagia for nine years when she first presented. The radioscopic and endoscopic findings were those of cardiospasm and her symptoms were relieved by a single dilatation. She returned three years later, still swallowing normally but with hoarseness of voice. The right vocal cord was immobile and lay in the cadaveric position and she was shown radioscopically and endoscopically to have a carcinoma, histologically squamous, in the middle third of the œsophagus. The tumour was irradiated. Six months later she died from metastases.

In 12 patients symptoms persist after dilatation of the cardia and the radioscopic appearances are still those of cardiospasm. These patients do, however, claim improvement and all have gained weight. They regurgitate less frequently but they eat slowly and are aware of food sticking at levels varying from the xiphisternum to the suprasternal notch. In these patients, as in many other patients investigated for dysphagia, the relationship between the level of the obstruction and the level at which dysphagia is experienced is inconsistent. It is common for patients to site the level of any œsophageal obstruction as proximal to the endoscopic level; the level of the obstruction as given by the patient with cardiospasm has not corresponded with the upper limit of retained food in the œsophagus (Allison, 1953), where this has been appreciable as a level radiographically; and a few patients have complained of dysphagia at a level distal to the site of an organic or functional obstruction.

If a history were being taken from these 12 patients for the first time, they would be regarded as having cardiospasm and as being in need of treatment. Radioscopically the œsophagus in some of them is reduced in calibre but barium is still held up, the column of barium varying in height from the level of the lower pulmonary vein to the aortic arch. Peristalsis is deficient and inco-ordinated and only one has air in the gastric fundus. These patients are all suitable candidates for cardiomyotomy, but for a variety of reasons they have not been submitted to operation. Six are unwilling to undergo any further treatment because they are sufficiently impressed with the improvement in their symptoms to regard themselves as relieved of dysphagia. Two are 74 years of age and are able to swallow reasonably well with occasional dilatation, making the additional hazard of thoracotomy at this advanced age unjustifiable. Two have recently left the region and are untraceable. Two have very gross œsophageal dilatation, the œsophagus occupying most of the right hemithorax, with œsophageal redundancy and acute kinking at the distal end. Heller's operation is unlikely to relieve their symptoms just as it failed to relieve dysphagia in the single patient in this series with a sigmoid œsophagus in whom cardiomyotomy was undertaken.

Therefore, of 28 patients with cardiospasm treated only by hydrostatic dilatation, 14 are regarded as having satisfactory clinical and radioscopic results, 2 were relieved of dysphagia but subsequently died, and 12 failed to derive prolonged and objectively satisfactory improvement.

THE TECHNIQUE OF CARDIOMYOTOMY

Heller's operation was undertaken in 1 patient through the abdomen and in 25 patients using the following standard transthoracic technique. The cardia and distal œsophagus were approached through the left chest, through a thoracotomy in relation to the bed of the unresected 9th rib, without transgression of the costal margin. The œsophagus was mobilized in the mediastinum from the hiatus œsophageus to the level of the lower pulmonary vein, by incising the mediastinal pleura and hooking the œsophagus out of its bed with a finger. Aortic branches to the œsophagus were rarely encountered. The œsophagus was always bulky and cylindrical, rather than strap-like, as it is when stretched over the finger in a normal subject. Its external diameter varied from patient to patient, but it was always larger than normal. So that the peritoneal cavity could be explored, the diaphragm was detached peripherally from the chest wall, forwards as far as the pericardium and backwards to the upper pole of the kidney, leaving a 1 cm. cuff of diaphragm on the chest wall (le Roux, 1961). This transdiaphragmatic entrance into the peritoneal cavity can be securely closed and leaves a normally functioning diaphragm. The abdomen was explored through the diaphragmatic incision with particular reference to the exclusion of carcinoma in the gastric fundus and to the demonstration of peptic ulceration in the duodenum.

The œsophageal muscle was incised in the length of the œsophagus, exposing mucous membrane proximally to about the level of the lower pulmonary vein and distally to the point where the œsophageal muscle was no longer hypertrophied. The proximal

limit of the muscular incision was arbitrary. All that was considered necessary was to incise hypertrophied muscle, and to ensure that this had been properly done the incision was carried proximally for an appreciable distance. This incision through the muscle was now continued distally, exposing mucosa of the chest showed the diaphragm to lie at a normal level. Radioscopy of patients in whom this incision has been made has demonstrated normal diaphragmatic movement up to ten years after thoracotomy. This approach through the diaphragm has been used about 300 times in operating on a variety of benign

FIG. 732.—The film on the right shows diminution in œsophageal calibre several years after relief of dysphagia by cardiomyotomy in a patient with cardiospasm. The radioscopic appearances before treatment are recorded on the films on the left. The radiograph on the right was made during the swallowing of 6 fl. oz. of barium and there was no barium in the œsophagus 90 sec. after this volume of barium had been drunk.

at the cardia, and for 2–3 cm. on the anterior gastric wall. To achieve the caudal limit of the incision through muscle the stomach was delivered into the chest through the hiatus œsophageus by gentle traction on the œsophagus. Care was taken to avoid disrupting the mechanism of the hiatus, either by incisions of the diaphragm or by unnecessary disturbance of the pleural and peritoneal reflections. If the stomach was not easily delivered through the hiatus the caudal limit of the incision through muscle on the anterior gastric wall was completed from below the diaphragm.

The intention was not to open mucosa; but in 8 of 25 patients submitted to transthoracic Heller's operation the mucosa was opened. Where this had been done the opportunity was taken of inspecting the mucosal aspect of the cardia to ensure that there was no other cause for obstruction. Where the mucosa had not been opened the anterior gastric wall was invaginated over two fingers from below the diaphragm through the cardia to ensure that the cardia was widely opened. Mucosa was closed transversely if it had been opened; the edges of the mediastinal pleura were approximated. The diaphragm was then re-attached peripherally. The pleural space was drained and the chest wall was closed.

Peripheral detachment of the diaphragm does not disturb its function. In a few patients light anæsthesia and loss of action of the relaxant used permitted observation of normal diaphragmatic function during closure of the diaphragm. No patient has been examined radioscopically earlier than eight days after this approach, but at this stage after operation the diaphragm moves in the normal direction and over a normal range both on quiet respiration and on sniffing. During the first eight post-operative days plain films

distal œsophageal and proximal gastric lesions and has been complicated only once, by herniation of peritoneal contents through an inadequately re-attached diaphragm. This transdiaphragmatic hernia was recognized a year after the original operation. The technique of peripheral detachment of the diaphragm was designed by Logan.

THE RESULTS OF HELLER'S OPERATION

Fourteen of the 26 patients submitted to Heller's operation are symptom-free three to twelve years after operation. Radioscopically these patients have been shown not to have any hold-up of barium at the cardia and the œsophagus is of normal calibre (*Fig.* 732). In all of them there is a gas bubble in the gastric fundus. In none of them is œsophageal peristalsis normal and in most there is radioscopic evidence of gross intermittent irregular œsophageal spasm. Five patients in addition to the 14 already mentioned have the same radioscopic features after cardiomyotomy as those described above, but these 5 patients still complain of intermittent dysphagia. In all 5 the œsophagoscopic appearances are normal; there is no residue within the œsophageal lumen and the distal end of the long adult Negus œsophagoscope can be introduced into the stomach. It may be that in these patients the symptom of dysphagia is a subjective reflection of œsophageal spasm. In 4 of these 5 patients œsophagoscopy has relieved symptoms for three to six months and dysphagia has not recurred at the time of writing. The fifth patient still complains of the sensation of food sticking at the level of the suprasternal notch once or twice a week. All 19 patients with the radioscopic appearances of relief of œsophageal obstruction after cardiomyotomy gained weight after the operation.

One patient, a girl now aged 16 years, who presented with severe dysphagia, claims to be relieved of all symptoms and gained 2 st. in weight during the first three months after Heller's operation. Radioscopically, two years after operation the first mouthfuls of barium pass rapidly into the stomach and then a column of barium collects to reach nearly to the level of the pulmonary hilum, remaining there for at least three minutes after 6 fl. oz. of barium have been drunk (*Fig.* 733). The œsophagus is reduced in calibre in comparison with the appearances before cardiomyotomy.

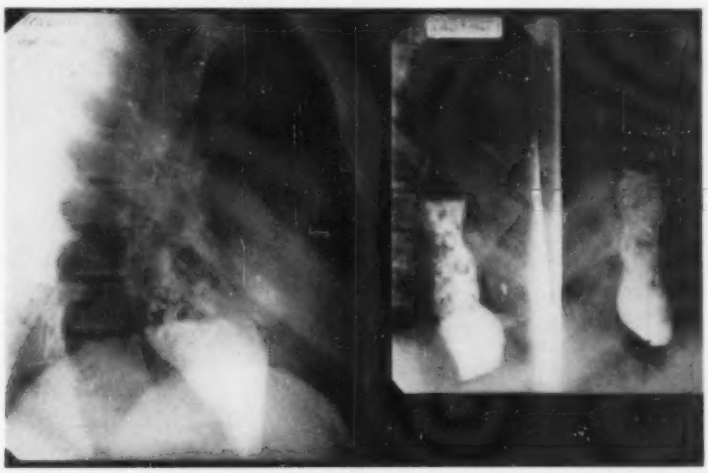


FIG. 733.—The patient whose radiographs are shown here was relieved of dysphagia and gained weight after Heller's operation. The film on the right was made two years after cardiomyotomy, the radioscopic result of which is regarded as unsatisfactory because the œsophageal lumen is little reduced in calibre and barium is retained in the œsophagus 3½ min. after 6 fl. oz. have been drunk.

Four patients submitted to Heller's operation still complain of dysphagia, although less severe than before operation, and still have the radioscopic features of cardiospasm—œsophageal dilatation and the retention of a column of barium for several minutes in the œsophagus, in addition to abnormal, inco-ordinated peristalsis. In 2 of these 4 patients dilatation of the cardia with the hydrostatic dilator has relieved dysphagia and improved the radioscopic appearances, but these patients have been observed for only six months after dilatation. In the third of these 4 patients with an unsatisfactory clinical and radioscopic result from Heller's operation there is an acute kink at the distal end of the œsophagus (*Fig.* 734), and endoscopy, with introduction of the tip of the endoscope into the stomach, does not relieve dysphagia. In this patient some form of resection seems inevitable, keeping in mind the bad results which attend many resections undertaken in patients with cardiospasm (Barrett and Franklin, 1949) and in whom an œsophagogastric anastomosis has been made. The fourth patient has been observed for two years following cardiomyotomy; the œsophagus has diminished considerably in calibre, having originally been visible on radiographs of the chest, but he has lost weight after an initial increase, and dysphagia is relieved for only short intervals by dilatation. It is proposed to submit this patient to a second Heller's procedure.

Two patients submitted to Heller's operation are dead. One man died six months after cardiomyotomy, dysphagia having been relieved by this procedure. He presented elsewhere with melæna, and was submitted to distal gastrectomy for a bleeding duodenal ulcer. This operation was complicated by gastric

FIG. 734.—A post-operative film from a patient in whom cardiomyotomy was unsuccessful in the relief of symptoms. The reason for failure is the persistence of œsophageal redundancy and the sigmoid curve at the distal end of the œsophagus. Barium overflows into the stomach only when this redundant sump is filled. The radioscopic appearances are quite different from untreated cardiospasm because there is a continuous flow of barium into the stomach once the sump has been filled.

FIG. 735.—The opacity parallel to the right border of the heart below the pulmonary hilum is shown, on the film on the right, to be the œsophagus when a tube with an opaque tip has been swallowed. The dilated œsophagus in cardiospasm usually presents first on plain films well above the right pulmonary hilum.

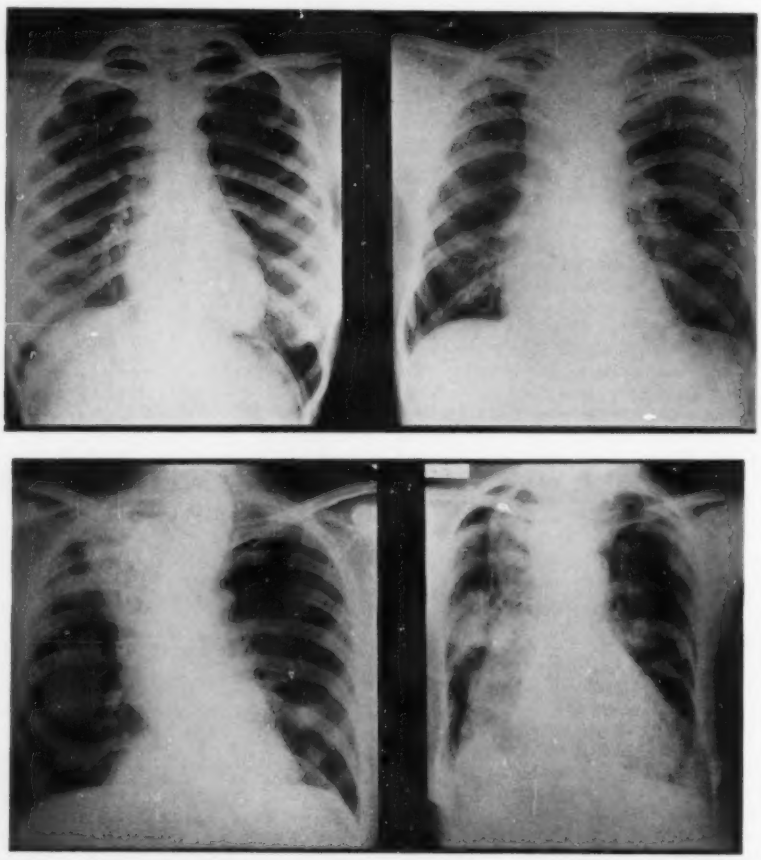


FIG. 736.—Postero-anterior views of the chest in 4 patients with cardiospasm showing variations in the shadow made by the dilated œsophagus. Pulmonary opacities, the consequence of aspiration, are seen in the two lower films.

stasis, and after a subsequent surgical procedure to relieve stasis he died from respiratory infection. The other patient who died after Heller's operation was a woman of 75, the oldest in the series, and one of the first treated in this unit, in 1948. Her history was of dysphagia from the age of 16 years, and she attributed the onset of symptoms to the death of a brother. When first seen the patient was ill from inhalation pneumonia, cachectic, and with total dysphagia. The œsophagus was visible on postero-anterior chest radiographs. The cardia could not be located at œsophagoscopy, and the appearances of the œsophageal mucosa were those of acute hæmorrhagic inflammation. The regurgitated œsophageal contents had been heavily blood-stained for several days. It was decided that Heller's operation was the only procedure which would relieve œsophageal stasis, regurgitation, and inhalation, and at the same time facilitate hydration and nutrition. Cardiomyotomy was undertaken through the abdomen; she died from pulmonary infection on the fourth postoperative day.

Thus of the 26 patients submitted to cardiomyotomy, 1 died from operation; 1 died six months later having swallowed normally during this period; 4 have been inadequately relieved of their symptoms; 1, while symptom-free, has an unsatisfactory radioscopic result; 5 with a satisfactory radioscopic result have occasional dysphagia; and 14 are symptom-free and have satisfactory radioscopic results.

Unusual Cases.—A man of 59 presented with the complaint of dysphagia for two months. The plain film of the chest was typical of gross œsophageal dilatation associated with long-standing cardiospasm. Œsophagoscopically he was found to have a carcinoma at 39 cm. from the upper alveolus, with the other endoscopic features associated with cardiospasm. Œsophagogastrectomy was undertaken for adenocarcinoma in 1959 and he remains well at the present time. Glands were not invaded by tumour.

Two patients were submitted to Heller's operation through a left thoracotomy after a transabdominal Heller's operation undertaken elsewhere had failed to relieve symptoms. The appearances at thoracotomy were those found routinely in cardiospasm and the conclusion was that the first operation had been inadequate in so far as the incision through muscle could not have extended sufficiently far proximally to include the cardia.

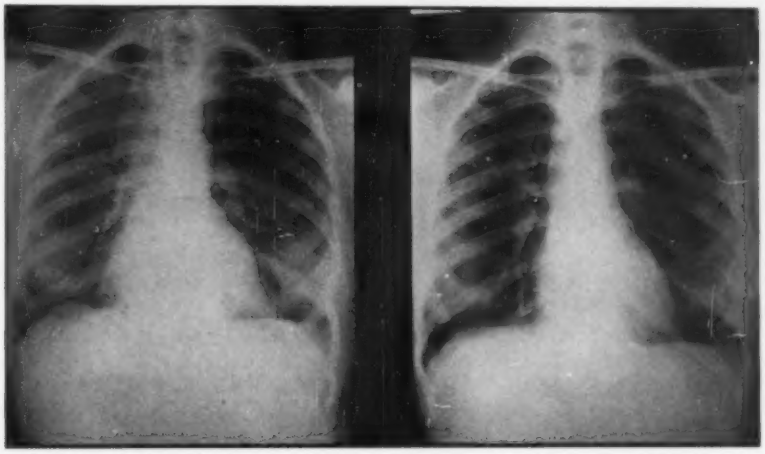
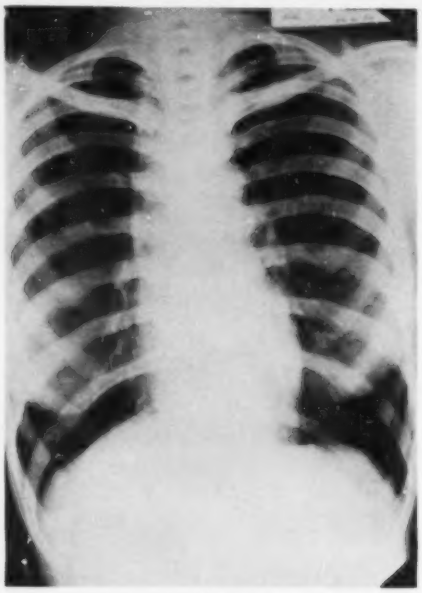


FIG. 737.—The shadow of the dilated œsophagus shown in the film on the left had disappeared in this patient the day after œsophageal lavage. The cardia could not be found at œsophagoscopy so that disappearance of the shadow could not be attributed to relief of distal obstruction.

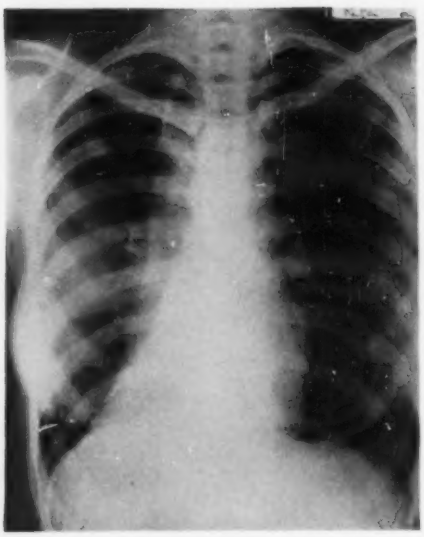
FIG. 738.—Gross œsophageal dilatation with forward displacement of the trachea.

At a time when it was part of the routine in this Unit to make a biopsy at œsophagoscopy from the distal end of the œsophagus in order to establish the range of normal in biopsy material from this

site, 2 patients with the radioscopic and endoscopic appearances of cardiospasm were reported on examination of a biopsy specimen to have histological evidence of squamous carcinoma in the distal



A



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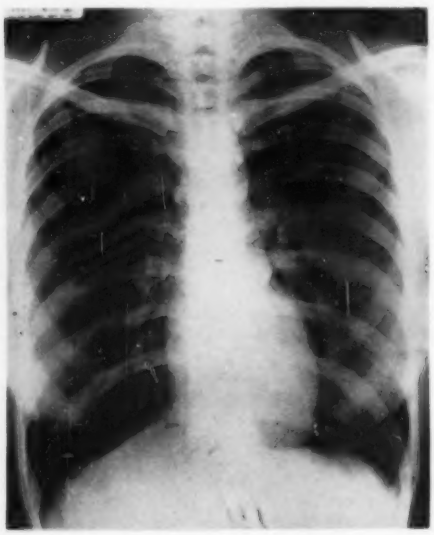

C

FIG. 739.—Radiographs of the chest in a young woman with cardiospasm showing: A, The œsophagus visible to the right of the mediastinum; B, Some months later, atelectasis of the right lower lobe; C, A normal radiograph four years after Heller's operation.

œsophagus. There was no clinical, radioscopic, or endoscopic reason for suspecting these patients of having a carcinoma, but, in view of the histological interpretation, it was regarded as unsafe to adhere to the criterion of the trial—i.e., the assessment of the results of dilatation before undertaking Heller's operation—and these patients were therefore submitted to left thoracotomy. Exploration of the mediastinum and of the abdomen confirmed that these patients did not have a carcinoma: the surgical findings were those of cardiospasm: cardiomyotomy was undertaken and both patients are well, eight and ten years respectively, after operation.

Of a series of 70 patients submitted to resection of a peptic stricture of the œsophagus, in 5 tissue taken from an œsophageal stricture, endoscopically benign, was reported to show the changes of carcinoma. No tumour was found in the specimen resected from any of these 5 cases. For the experienced endoscopist the appearances at œsophagoscopy in conjunction with the radioscopic findings with barium are diagnostic. If there is any doubt regarding the cause of distal organic dysphagia, the patient must in any event be submitted to thoraco-laparotomy. It is therefore important to be selective in making a biopsy from the cardia at œsophagoscopy since the interpretation of such a biopsy even in expert hands is apparently unreliable.

THE RADIOGRAPHIC FEATURES OF CARDIOSPASM

The radiological diagnosis of cardiospasm in the established case is rarely difficult. Gross œsophageal dilatation in association with smooth narrowing and delay in the passage of barium at the hiatus are characteristic. In a patient presenting before dilatation of the œsophagus has developed the only radioscopic abnormality may be persistent failure of the cardia to open. If such a patient swallows a

small bread crust soaked with barium, this will be seen to lodge at the cardia. In early cases without œsophageal dilatation the presence of abnormal peristalsis in the œsophagus (Johnstone, 1956) supports the diagnosis of cardiospasm, and the (Sleisinger, Steinberg, and Almy, 1953) has an action like acetylcholine when injected intravenously, and in cardiospasm increases intraluminal pressure by inducing spasm of the œsophagus. This response is specific, and, in accordance with Cannon's law of

FIG. 740.—There is a superficial resemblance between the films of the barium-filled œsophagus in a patient with a carcinoma at the cardia (*left*) and the film of the œsophagus in a patient with cardiospasm (*right*). The filling defect at the distal end of the œsophagus on the left precludes the diagnosis of cardiospasm.

response to Buscopan (hyoscine-N-butylbromide) is conclusive (Wright, 1960).

The radiologist has the opportunity of recognizing radiographically the apparently symptomless case of cardiospasm in whom the mediastinal shadow on plain films is increased, usually to the right (*Figs.* 735, 736). If the lateral border of the mediastinal shadow crosses the pulmonary hilum the diagnosis can be suggested with confidence. If the mediastinal shadow changes shape from one radiograph to another, or contains a fluid level, the diagnosis is established. The radiographic shadow of a dilated œsophagus may disappear very soon after lavage of the œsophagus, alone or combined with dilatation of the cardia (*Fig.* 737). The dilated œsophagus does occasionally prolapse to the left (Allison, 1953). In lateral radiographs a mottled opacity, the consequence of food residue in the œsophagus, is often visible behind the trachea, displacing this forwards (*Fig.* 738). Œsophageal obstruction from carcinoma may occasionally produce this appearance. In 13 of the 55 patients reviewed in this paper the œsophagus was visible to the right of the mediastinum on plain films of the chest. Three of these 13 patients were found during routine radiographic survey and in 7 of them Heller's operation was undertaken. Respiratory symptoms are common in cardiospasm, and in 15 patients in this series there was radiographic evidence of pulmonary infection—patchy pneumonic changes in 9 and lobar collapse in 6 (*Fig.* 739). The pulmonary lesions of cardiospasm are the consequence of aspiration.

In the radioscopic investigation of cardiospasm certain tests have been devised, based on the response of the abnormal œsophagus to drugs. The oldest of these is the octyl nitrate test; this drug relaxes smooth muscle. Relaxation is transient and the drug has unpleasant side-effects, discouraging its use. Mecholyl

FIG. 741.—The film on the left shows the appearances of the barium-filled œsophagus in a patient with cardiospasm. The film on the right was made shortly after this patient had been given Buscopan. The œsophagus has emptied rapidly, by gravity, and a gas bubble has appeared in the gastric fundus.

denervation (Cannon and Rosenblueth, 1936), implies destruction of Auerbach's cells and plexus. In the cases reported in this series these investigations have not been used. The effect of Buscopan on the cardia in cardiospasm has been recorded in detail elsewhere (Wright, 1960), and this drug has been used on some of the patients in this series. Carcinoma at the cardia may simulate cardiospasm (*Fig.* 740) and

carcinoma of the œsophagus may complicate cardiospasm. Abnormal œsophageal peristalsis and the absence of a gas bubble in the fundus may help in differentiating cardiospasm from carcinoma at the cardia, but these features are not conclusive in themselves. Where differentiation is in doubt, radioscopically, and in early cases of cardiospasm without œsophageal dilatation, Buscopan has been shown to be a useful aid in diagnosis. In cardiospasm this drug stimulates opening of the cardia with strikingly rapid emptying by gravity of the barium-filled œsophagus, sometimes with the appearance of a gas bubble in the gastric fundus (*Fig.* 741). In the patient with carcinoma at the cardia this drug has no effect in hastening œsophageal emptying.

In the assessment of the effects of Heller's operation and of hydrostatic dilatation of the cardia in cardiospasm certain features have recurred with significant frequency. The routine in the radioscopic assessment of these patients was as follows:—

The patient in the erect position was given a cup containing 6 fl. oz. of a 50/50 suspension of barium and was asked to swallow this as quickly as possible.

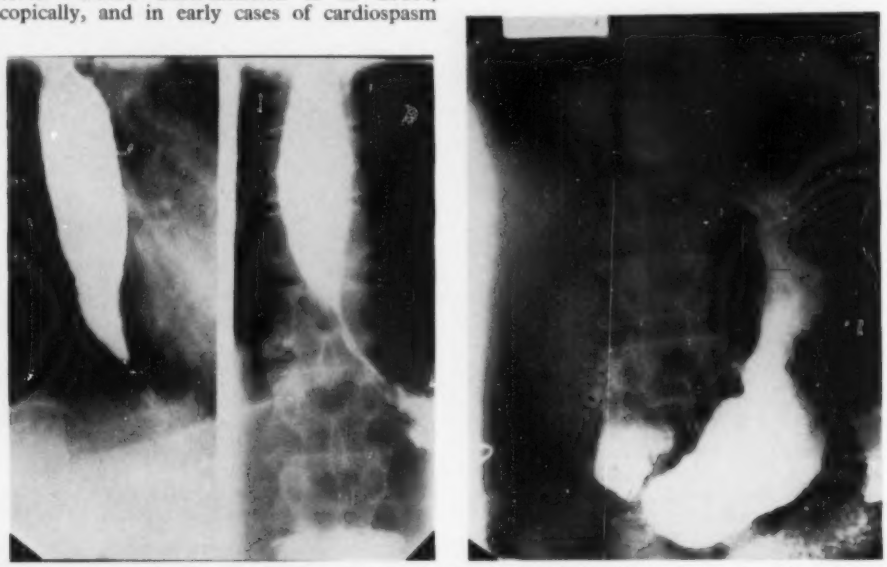

FIG. 742.—The patient whose films are shown claimed relief of dysphagia following hydrostatic dilatation of the cardia. Although the calibre of the œsophagus is still abnormally large on the film on the left, there was no hold-up at the cardia and all the barium in the œsophagus had entered the stomach, as shown on the film on the right, within 3 min. of drinking 6 fl. oz. of a barium suspension.

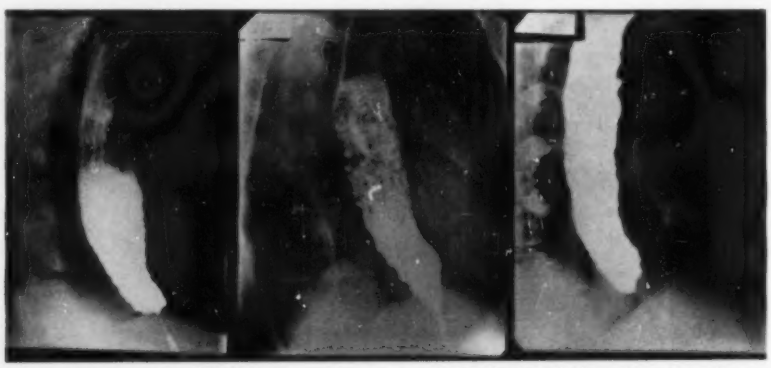

FIG. 743.—Three examples of ripples or corrugations in the wall of the barium-filled œsophagus in cardiospasm.

When the barium had been swallowed three films of the œsophagus were taken—two oblique views and a postero-anterior view. Thereafter, 3 min. from the start of swallowing barium, a film of the stomach was taken (*Fig.* 742) and then, as quickly as possible, another of the œsophagus. This gave a permanent record of the size of the œsophagus and of the type of œsophageal peristalsis, of barium in the stomach within 3 min. of deglutition, and of the quantity of barium retained in the œsophagus about 3½ min. after deglutition. This routine provided a reasonably

clear picture of successful or unsuccessful relief of obstruction which tallied fairly closely with the clinical assessment of the patients examined. However, in 5 patients who complained of dysphagia after Heller's operation there was no residue of barium in the œsophagus, which was of normal calibre, and 1 patient who denied dysphagia after Heller's operation still had significant hold-up at the cardia.

In a comparison of the post-operative appearances with those recorded pre-operatively, abnormal peristalsis and spasms of the œsophagus become very much more obvious after satisfactory opening of the cardia, and normal peristalsis is not seen. Radioscopically œsophageal spasms are quite dramatic, and during the period of post-operative observation—in some cases twelve years—no œsophagus has been seen to return to normal.

The abnormalities of œsophageal movement which persist after relief of distal œsophageal obstruction in cardiospasm may be of three varieties: ripples or corrugations on the margins of the œsophagus little different from the tertiary waves often seen before treatment (*Fig.* 743); intermittent concentric narrowing, as if the œsophagus had been gripped by a fist (*Fig.* 744); and intermittent diffuse spasm which may involve as much as a third of the œsophagus, usually the lower third (*Fig.* 745). These abnormalities of motility are very similar to those seen in corkscrew œsophagus, a common discovery during routine radioscopy, and for the most part symptomless. In corkscrew œsophagus, however, œsophageal pressure records indicate that there is a background of normal peristalsis, absent in cardiospasm (Ellis, Olsen, and Holman, 1958). The wideness of the œsophageal lumen in cardiospasm probably prevents the development of the appearance, common in corkscrew œsophagus, called pseudodiverticulosis.

In no case of cardiospasm in this series was there evidence of normal organized progressive peristaltic activity in the œsophagus, and gravity would appear to be the only means whereby the œsophagus is emptied. Barium is heavy and enters the stomach quickly if the obstruction at the cardia has been released. If the satisfactorily treated patient is placed in the supine position, barium lies in the œsophagus indefinitely. Air is light and must therefore, in the erect position, be propelled into the stomach by peristalsis. This may be the explanation of the small or

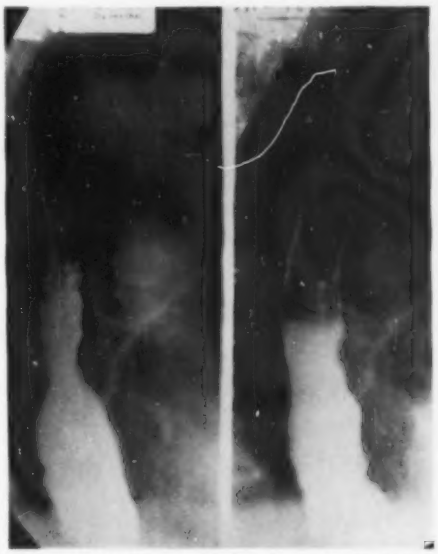

FIG. 744.—The film on the left shows œsophageal spasm of the type likened to an œsophagus gripped by a fist. The film on the right was taken very shortly afterwards and the spasm is no longer present. These films record the appearances at radioscopy after cardiomyotomy in the patient whose pre-operative film is shown on the right of *Fig.* 740.

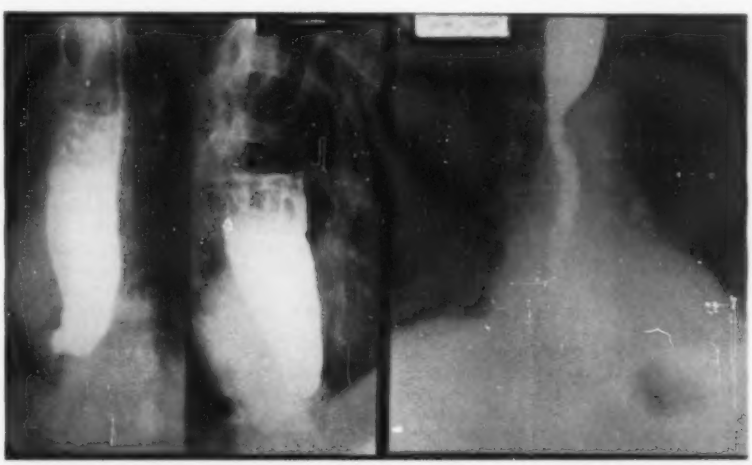

FIG. 745.—The film on the left shows the appearances in a patient with cardiospasm before cardiomyotomy. In the film on the right made some months after cardiomyotomy diffuse spasm involves the lower third of the œsophagus. Barium was not retained in this œsophagus for an appreciable length of time, and when the œsophagus was not in spasm its calibre was normal. This patient still complained of occasional dysphagia and this was attributed to œsophageal spasm.

absent gas bubble in the gastric fundus in cardiospasm as against the normal gas bubble in œsophageal carcinoma, with a similar degree of obstruction and dilatation, but with little disorganization of peristalsis. The return of the fundic gas bubble in many treated cases of cardiospasm can be explained either by air entering the stomach when the patient is in the recumbent position, without the help of œsophageal peristalsis, or by air being trapped in food during swallowing.

FIG. 746.—A grossly dilated sigmoid œsophagus.

In about one-third of the patients in this and in most other series, the œsophagus remains for practical purposes an inert bag which does not empty as rapidly as it is filled, with the result that food lies in an œsophageal sump for a variable length of time. If the œsophagus is redundant, with an S-bend just above the cardia, such a sump will be permanent, like the water-seal in the domestic water-closet. A sigmoid œsophagus was found in 3 patients in this series (*Figs.* 746, 747).

One further radioscopic observation made during the investigation of the cases in this series was the remarkably violent peristalsis which may be generated in the distal half of the œsophagus—so violent that barium which has already reached the most distal œsophageal segment may be forced back up the œsophagus to a level above the aortic arch. This phenomenon may explain the occurrence of post-operative dysphagia in those patients in whom satisfactory relief of distal œsophageal obstruction has been shown radioscopically. Five patients in this series with dysphagia after cardiomyotomy in whom there was no hold-up of barium, and in whom the œsophagus was of normal calibre, had particularly violent œsophageal motility radioscopically and in 3 this phenomenon of retrograde ejection of barium was observed.

In patients in whom dysphagia has persisted and with slow clearing of barium from the œsophagus the demonstration of gastro-œsophageal reflux after cardiomyotomy is usually impossible because the œsophagus is not empty. In 1 patient in this series—one of three who complained of post-operative heartburn—it was possible to demonstrate post-operative reflux and a small hiatus hernia. The muscle of the cardia can normally play very little part in the prevention of reflux, for if it did a successful Heller's operation would usually be followed by gastro-œsophageal reflux. Disturbance of the crural sling about the hiatus œsophageus is the likely explanation of the occurrence of reflux in this single instance. Reflux could not be demonstrated in the 2 other patients with heartburn and none of the 3 patients with heartburn had the œsophagoscopic changes of peptic œsophagitis. None of the 55 patients in this series was shown to have a hiatus hernia before treatment.

FIG. 747.—A sigmoid œsophagus with very much less dilatation than in the example shown in *Fig.* 746.

THE CLINICAL FEATURES OF CARDIOSPASM

These have been well documented in the past. Allison (1953) described the clinical features and Ellis (1960) outlined the natural history of the disease. No patient in the present series was found to have finger clubbing; apart from this all the accepted clinical features of the disease were encountered.

Disturbances of swallowing are commonly related to disturbances of psychological equilibrium. It is not known how real this association is in cardiospasm, but there are well-established examples of disturbances of œsophageal motility the consequence of pyschological trauma. In the records of the Edinburgh Thoracic Unit there are many patients who presented

with dysphagia apparently initiated by psychological stress, in whom no cause for dysphagia was found, and all of whom were relieved of their symptoms with the passage of time. One of the more clearly defined examples of psychogenic dysphagia is the instance of the miner, aged 56 years, who escaped physical injury when an explosive was prematurely detonated. He was considerably shaken by this experience but continued work for the remainder of his shift. He returned home; the first few mouthfuls of his evening meal were felt to pass down with a little discomfort, and then a bolus of normally chewed meat impacted, according to the patient, at the level of the xiphisternum. Thereafter he was in considerable pain and had total dysphagia, including dysphagia for liquids. Eighteen hours after the onset of dysphagia he was examined with the œsophagoscope. A large quantity of food was removed from the distal œsophagus, which thereafter looked normal. Following œsophagoscopy he swallowed normally, the radioscopic appearances were normal the following day, and again six months later, and the man remains well, without recurrence of dysphagia, five years after the episode.

Organic lesions of the œsophagus occasionally have their origin, according to the patient, in an episode of psychological trauma—for example, a patient shown by investigation to have an œsophageal carcinoma may give a history of the initial episode of dysphagia occurring during psychological stress, and however real this relationship may be for the patient the growth of the carcinoma cannot be attributed to psychological trauma. It is, therefore, possible that the common relationship between psychological trauma and dysphagia in cardiospasm has the same chance association, and that the known influence of psychic stress on œsophageal motility does no more than bring to light the established œsophageal lesion. The only justification for relating the onset of cardiospasm to emotional stress is the frequency of the relationship. In the records of the 55 patients with cardiospasm reported in this series reference to psychological stress is common. On reading these histories, it is not possible to decide whether the patients volunteered this relationship or whether such information was wrested from them by interrogators who knew of the reputed relationship between emotion and cardiospasm. The taking of fresh histories from treated patients is unsatisfactory, but many of them still associate the origin of dysphagia with an episode of stress. For example, the death of a relative was blamed in 4 cases; in 3, symptoms dated from starting work for the first time at the age of 16 or 17; dysphagia in a relative precipitated symptoms in 2 cases and escape from injury in a motor-car accident in 1 case; symptoms in 5 females began at the time o puberty and were attributed by all of them to the stress associated with written or oral examinations at school. Three male patients were tellers in banks—an occupation reputedly associated with considerable mental strain. A fourth patient was also a banker—a manager of a large branch—but he became aware of dysphagia only after he retired at the age of 65 years. The fact that the more acute episodes of psychological stress blamed as a precipitating cause of dysphagia by some of these patients are common to most people at some time is no argument against such stress precipitating symptoms in the susceptible subject. Many patients who did not offer a clear history of precipitating psychic trauma were of a type that Allison (1950) called 'odd'. Six patients were, however, at least to observers untrained in psychiatry, the most normal phlegmatic individuals imaginable. Three of these 6 were found to have a grossly dilated œsophagus when they were X-rayed during a mass radiography campaign, and all 3 denied symptoms. When œsophageal stasis had been relieved in these 3 patients by cardiomyotomy, they all volunteered that swallowing post-operatively was a new experience for them and quite different from that to which they were accustomed.

In the patient with severe cardiospasm detailed radioscopic examination of the stomach and duodenum may be difficult because insufficient barium passes through the cardia. In 5 patients in this series it was, however, possible to demonstrate duodenal ulceration. In a further 2 patients dysphagia, the consequence of cardiospasm, was appreciated for the first time during convalescence from distal gastrectomy for a duodenal ulcer, and in yet another patient, previously mentioned, death was the consequence of operations for a bleeding duodenal ulcer six months after Heller's operation. The psychological build of the patient may be the common factor in the development of cardiospasm and duodenal ulcer, but a more likely relationship is that cardiospasm is the clinical corollary of sham-feeding experiments in dogs. The sight, smell, and taste of food stimulate gastric secretion and the duodenum is exposed to these secretions undiluted by food which is retained in the œsophagus (Spurrell, 1953).

Thirty-seven patients included in their story the complaint of loss of weight; 16 of these patients underwent Heller's operation. The 54 survivors of treatment all gained weight and a gain of 2–4 st. was common within three months of either dilatation of the cardia or cardiomyotomy. Thirty-four patients gave a history of recurring respiratory infections and 19 of these underwent Heller's operation.

THE ASSOCIATION BETWEEN CARDIOSPASM AND CARCINOMA OF THE ŒSOPHAGUS

The incidence of œsophageal carcinoma complicating cardiospasm varies in different series. Two of the 55 patients reported in this series developed a carcinoma; a female patient died from a squamous carcinoma of the middle third of the œsophagus and a male with an adenocarcinoma at the cardia was submitted to resection. Ellis (1960) found that 6 of 24 patients with cardiospasm who died had in addition an œsophageal carcinoma. Lawrance and Shoesmith (1959) reviewed 118 patients with cardiospasm; 17 of these had died, 3 from œsophageal carcinoma. Groves (1956), Potter, Rasmussen, and Best (1956), and Peyman (1959) have reported cases of carcinoma of the œsophagus complicating cardiospasm, one of them complicated by hypertrophic pulmonary osteoarthropathy. The literature relating to the incidence of carcinoma of the œsophagus in patients with cardiospasm is reviewed by these authors, and this incidence is usually given as considerably less than 10 per cent, but Belsey (1958) reported a 12 per cent incidence. There is little doubt that patients with

cardiospasm are exposed to a greater risk of developing œsophageal carcinoma than is the population at large. The accepted explanation for this is the exposure of the œsophageal mucosa to chronic irritation by retained food. One of the two examples of carcinoma complicating cardiospasm reported in this series had been relieved of dysphagia by dilatation of the cardia three years before presenting with an already disseminated œsophageal carcinoma. In this patient there was no significant hold-up of barium at the cardia when she was examined radioscopically three years after dilatation. Relief of distal œsophageal obstruction may therefore not necessarily be an assurance against the subsequent development of a carcinoma.

In patients with cardiospasm the diagnosis of carcinoma of the œsophagus is likely to be delayed because they are accustomed to dysphagia; and because the radioscopic demonstration of carcinoma in the already dilated œsophagus is difficult. The carcinoma growing in a dilated œsophagus must achieve a considerable size before obstructing significantly the lumen of the œsophagus. Such a tumour is likely to have been present for a long time and therefore likely to have disseminated.

PATHOGENESIS OF CARDIOSPASM

The aetiology of cardiospasm is not known, its pathogenesis is therefore obscure, and the pathological changes in the established case may be either primary or secondary. Mikulicz (1904) and Hurst and Rake (1929) believed that the primary fault lay in the cardia, an area notoriously difficult of both anatomical and physiological definition. Barrett (1953), reflecting on the aetiology of cardiospasm, was able to discard some of the theories offered to explain the resistance to the onward passage of food at the cardia. In particular, the so-called 'pinch-cock mechanism' of the diaphragm as a cause of obstruction at the cardia cannot operate because the obstructed cardia in gross cardiospasm may lie several centimetres below the diaphragm, and there are many reported examples of cardiospasm occurring in association with hiatus hernia, the cardia lying proximal to the diaphragm (Euphrat and Delmonico, 1957; Olsen, Holman, and Anderson 1953; Palmer, 1956). Marchand (1955) and Torrance (1958) have contributed to the knowledge of the function of the distal œsophagus and its 'sphincter', but the mechanisms which co-ordinate peristalsis are unknown, and, while peristalsis can be disturbed in many ways, it is not understood how such inco-ordination is brought about. The absence of normally co-ordinated peristalsis in the œsophagus in cardiospasm is well recognized (Butin, Olsen, Moersch, and Cade, 1953; Johnstone, 1956). Pressure recordings (Ellis and others, 1958) and radioscopic findings confirm that normal peristalsis is not restored by relief of distal œsophageal obstruction.

Alnor (1958) has been able to reproduce in dogs the clinical, radioscopic, morbid anatomical, and histological features of the established case of cardiospasm by freezing the external surface of the cardia with carbon-dioxide snow. In these experimental animals there was no mucosal damage. These experimental findings are cited as evidence against spasm playing any part in the disease, but there is unequivocal evidence of spasm in the untreated case by the response to Buscopan, and, in the patient in whom distal œsophageal obstruction has been relieved, œsophageal spasm is a constant finding. The disease cardiospasm is not confined to the cardia, and is probably primarily a neuro-muscular disorder involving the smooth muscle of the distal two-thirds of the œsophagus.

DISCUSSION

The results of treatment in this series of 55 patients are little different from the recorded results of others (Hawthorn, Frobese, and Nemir, 1956; Herron, Thomas, and Merendino, 1957; Tuttle, Crowley, and Barrett, 1958; Acheson and Hadley, 1958; Lawrance and Shoesmith, 1959). Treatment, whether by hydrostatic dilatation of the cardia or by cardiomyotomy, will relieve dysphagia in 60–70 per cent of patients. Normal œsophageal peristalsis is never restored, and relief of œsophageal obstruction with reduction in the calibre of the œsophagus uncovers, radioscopically, grossly disordered œsophageal motility. No satisfactory form of treatment has been found for the patient with the grossly dilated œsophagus the distal end of which is kinked.

SUMMARY

Fifty-five cases of cardiospasm are presented. Twenty-eight were treated by hydrostatic dilatation of the cardia and 26 by cardiomyotomy. A coincident carcinoma in 1 case necessitated resection of the distal œsophagus and proximal stomach. In this series relief of dysphagia is not necessarily associated with radioscopic improvement, and conclusive radioscopic evidence of relief of distal œsophageal obstruction is not necessarily associated with relief of dysphagia. The radioscopic features of the disease and the changes induced by cardiomyotomy and by dilatation of the cardia on these features are presented in detail.

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SPONTANEOUS RUPTURE OF THE MIDDLE THIRD OF THE ŒSOPHAGUS

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SPONTANEOUS rupture of the œsophagus is a rare condition and carries a high mortality. Barrett (1947) reported the first success following early operative repair of the tear, and all recent authors agree with his view that early diagnosis and repair offer the best chance of cure (Ware, Schnider, and Davies, 1952; Moynihan, 1954; Derbes and Mitchell, 1956; Anderson, 1957; Pate, Hughes, and Patton, 1958). However, early diagnosis is not always easy, and many cases are still missed. The treatment of the few patients who survive the initial catastrophe is not so clear-cut, and this will be discussed.

In the majority of published cases the rupture has been in the lower œsophagus, just above the diaphragm, usually on the left posterolateral aspect. One case of rupture of the cervical œsophagus has been described (Russell, 1953), and three of the middle third of the œsophagus (Fitz, 1877; Allen, quoted by Terracol, 1938; Wachtel and Genkins, 1955). A further case of spontaneous rupture of the middle third of the œsophagus is reported here.

CASE REPORT

Mrs. C. Q., aged 73 years, was admitted to the Ipswich and East Suffolk Hospital on March 6, 1959, with a diagnosis of perforated peptic ulcer. For one month she had suffered from severe indigestion similar to attacks she experienced many years previously when a diagnosis of peptic ulcer had been made.

Two days before admission she developed frequency of micturition, accompanied by dysuria and hæmaturia. Her doctor prescribed liberal fluids, potassium citrate, and chloramphenicol 250 mg. q.d.s. Although the symptoms of cystitis cleared rapidly on this régime, she felt unwell and was only able to take a light diet.

On the day of admission she drank a cup of beef tea, and almost immediately vomited it. This was followed, within minutes, by a small hæmatemesis, and a severe, persistent pain in the epigastrium which radiated to the back, but not into the chest.

On examination 5½ hours later, she was still in great pain; lying quietly in bed; pale, but not cyanosed; temperature 97°; pulse 120, regular but of small volume; respirations 28; blood-pressure 130/80. There were no adventitious sounds in the chest, and air entry was normal. There was no evidence of cardiac failure. There was rigidity and tenderness all over the abdomen, but more particularly in the epigastrium; the liver dullness was not diminished, and there were no bowel sounds. Rectal examination revealed nothing abnormal. By gastric aspiration a small amount of clear thin brown fluid was obtained, which contained no macroscopic blood. A diagnosis of perforated peptic ulcer was made, but at laparotomy nothing abnormal was found.

Her condition after operation was satisfactory, and on the following morning she appeared to be very much better. An E.C.G. showed no evidence of coronary infarction, and the chest radiograph was reported as normal, although on subsequent review it showed the presence of mediastinal air (*Fig.* 748). The hæmoglobin

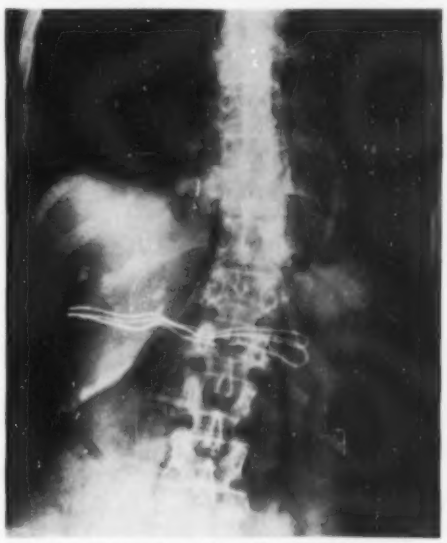

FIG. 748.—Radiograph 24 hours after the initial symptoms shows the presence of air in the mediastinum.

was 95 per cent, leucocytes 14,000 (80 per cent polymorphs, 19·5 per cent lymphocytes, and 0·5 per cent monocytes). However, she collapsed suddenly 36 hours after operation. The blood-pressure could not be recorded; the pulse was 140; the respirations were laboured and rattling in character; temperature 102° F.; and the limbs showed cyanosis. Subcutaneous emphysema was now noticed in the neck, and it was only then that a diagnosis of ruptured œsophagus was considered; there was no clinical evidence of a pleural effusion.

Noradrenaline and hydrocortisone were administered intravenously; antibiotic therapy in the form of penicillin 2 mega units t.d.s. and streptomycin 1 g. b.d. by intramuscular injection was started; and oxygen was given continuously. Gradually she responded to this treatment; the blood-pressure rose to between 100/60 and 140/85; the pulse became steady at ±100 with increased

volume. The pyrexia persisted, however, and later the antibiotic therapy was changed to intravenous terramycin 500 mg. b.d.

A second chest radiograph showed mediastinal and subcutaneous emphysema, and a small pleural effusion on the right side. The effusion was aspirated and replaced with soluble penicillin. The fluid was dark brown, thin, odourless, alkaline in reaction, and culture showed a *Staphylococcus aureus* sensitive to terramycin. The

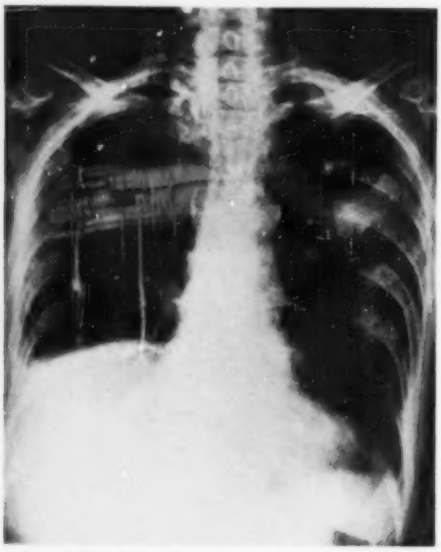

FIG. 749.—Radiograph on the 12th day after operation. Mediastinal and subcutaneous emphysema is obvious. A pleural effusion is seen on the right side, and an abscess is present in the left upper lobe.

diastase content was 250 U. (Wohlgmuth), which was interpreted as due to the presence of alimentary secretions.

The right pleural cavity was aspirated daily, and the fluid removed replaced first with soluble penicillin, and later with terramycin after the bacterial sensitivities had been obtained. The amount of fluid aspirated progressively diminished, and serial chest radiographs confirmed the initial favourable response; but the effusion never entirely cleared. Mediastinal emphysema was seen in all the subsequent films, but the subcutaneous emphysema absorbed rapidly.

Intravenous therapy was continued, and after 6 days small amounts of water, and then milk, were given through the Levine's tube. The fluids were gradually increased as no untoward effects were observed.

Although she appeared to be progressing well, there was still an occasional evening pyrexia, and the continual presence of air in the mediastinum was disturbing. On the 12th day the subcutaneous emphysema re-appeared in the neck, and extended down over the front of the chest. A radiograph showed an increase in the pleural effusion, and also a small abscess cavity in the left upper lobe (*Fig.* 749). All oral fluids were stopped. The latest sensitivity tests showed the *staphylococcus* in the pleural fluid to be sensitive to erythromycin only, and a course of this antibiotic was started. However, her condition deteriorated rapidly, and all resuscitative efforts failed.

At post-mortem a longitudinal 2-in. tear in the middle third of the œsophagus, on the right posterolateral aspect, was found. It communicated freely with the right pleural cavity. The œsophageal wall was pale and friable, but, apart from this, no other abnormalities were seen in the œsophagus. The right pleural space contained approximately one pint of thin pus with only a few friable adhesions, and there was no real attempt at localization of the pus. The right lower lobe was collapsed, and multiple small abscesses were present throughout the left lung. There were three small acute ulcers in the stomach and duodenum.

DISCUSSION

Site of Rupture.—Spontaneous rupture of the œsophagus usually follows the strain of violent vomiting. The mechanism is thought to be an overfilling of the œsophagus by stomach contents entering from below more rapidly than they can be expelled. Experimental support of this theory is given by McKenzie (1884). He used a healthy œsophagus removed from the cadaver immediately after death. One end of the organ was tied, and water introduced at the other under pressure. At a pressure of 7 lb. of water tears exactly akin to those found in spontaneous rupture were produced. With one exception, in eighteen experiments, a longitudinal tear occurred just above the cardia. The muscle coats suffered most severely.

His conclusions were that:—

1. Rupture by direct pressure within the œsophagus always took place in a longitudinal direction.
2. Rupture never occurred in the upper part of the œsophagus.
3. The mucosa appeared to offer greater resistance to the strain than the muscular layers.

He also considered the anatomy of the muscular coats important. The upper third is mainly striated muscle, and, when stimulated electrically, contracted forcibly. The lower third is predominantly plain muscle and electrical stimulation produces a slow peristalsis. The middle third is transitional, consisting of both striated and plain muscle. He considered that the lower third was less capable of resisting pressure.

Unfortunately, no microscopical examination was made in the present case, so that the histology of the muscle coat is not known. Possibly the anatomy and function of the muscle coat at various levels are important in determining the site of rupture. In the case of rupture of the cervical œsophagus described by Russell (1953), he thought that the underlying factor was a neuromuscular incoordination of the cricopharyngeus muscle and that of the upper œsophagus. It is interesting that his patient had a long history of dysphagia.

No local lesion was found at post-mortem to account for the unusual rupture. It occurred just below the constriction caused by the left main bronchus, and it was considered that there might have been a pre-existing diverticulum, but the tissues were so friable that no conclusions were reached. The tracheobronchial glands were normal and not adherent to the œsophageal wall.

Diagnosis.—Spontaneous rupture usually occurs in the age-group 35–55, often in people who eat and drink to excess. The onset is sudden with severe epigastric pain spreading to the back and lower chest after violent vomiting. The patient is extremely shocked, often cyanosed and dyspnœic, with variable chest signs, and tenderness and rigidity in the epigastrium. However, many cases do not present these classical signs. The condition has been

described in children (Benson and Penberthy, 1938; Moore and Murphey, 1948) and in older age-groups (Cliffton, 1949; Kernan, 1950; Clain and Heffernan, 1956). It may follow other types of straining effort such as defæcation (Whipple, 1927); asthma (Mitchell, Derbes, and Akenhead, 1955); weight-lifting (Griffith, 1952); or it may occur rarely without any straining effort (Mallam, Whitelock, and Robb-Smith, 1940). The condition most commonly confused with this lesion is a perforated peptic ulcer; but spontaneous pneumothorax, cardiac infarction, pancreatitis, and dissecting aneurysm must also be differentiated.

The present case was typical in that the severe pain followed immediately after a vomiting episode, but the patient had been having only a light diet. The pain did radiate to the back, but not into the chest, and it was concentrated mainly in the epigastrium. Although severely shocked, she was not cyanosed; there were no abnormal signs in the chest, and the abdominal signs were more widespread than is usually described.

Subcutaneous emphysema, pneumothorax, and hydropneumothorax do not always develop quickly, but there may be radiological changes in the chest before clinical signs are apparent, and a chest radiograph is a most valuable investigation. The early changes of spontaneous rupture of the œsophagus, such as mediastinal emphysema, broadening of the mediastinum, and blunting of the costophrenic angle, may be demonstrated (Priviteri and Gay, 1951). If a pleural effusion is present aspiration and examination of the fluid is indicated. This will show an acid reaction if stomach contents are present. The alkaline reaction obtained on this occasion was puzzling, due possibly to the escape through the tear of saliva rather than gastric contents. The diastase index is a useful investigation to confirm the presence of saliva or stomach contents.

It is important to know the site of rupture before operation. Christofordidis and Nelson (1957) suggest an X-ray using a water-soluble iodine medium rather than an oily substance as more likely to demonstrate a small leak.

The lack of chest signs in the present case may have been due to the abnormal site of rupture. When the lower unsupported part of the œsophagus ruptures the stomach contents are likely to escape directly into the pleural cavity, but the middle part is well supported, and with a small initial tear the effusion is probably localized to the posterior mediastinum at first. Further spread is delayed by stopping all oral fluids and keeping the stomach empty. The absence of subcutaneous emphysema and pleural effusion until the day after laparotomy supports this view. During this period, air tracked up the mediastinum to appear in the neck, and the mediastinal pleura on the right side was penetrated.

Treatment.—Successful results have been reported following conservative treatment of spontaneous rupture of the œsophagus (Anderson, 1952; Beal, 1949; Pate, Hughes, and Patton, 1958), but the illness is likely to be prolonged, and is complicated by empyemata, mediastinal abscesses, fistulæ, and perhaps suppurative pericarditis. The likelihood of ultimate survival, particularly in the elderly, is remote. Lynch (1949) and Pate and others (1958) report cases of successful delayed suture of the rupture after 65 and 49 days respectively.

Experience of the present case suggests that an attempt to repair the œsophagus once the diagnosis was made would have been the correct treatment. The favourable response to conservative treatment was misleading, and it was obvious at autopsy that no healing process had taken place in the œsophagus, nor was there any sign of localization of the pus in the pleura. Operative repair in the older age-groups must obviously be a hazardous undertaking, but successes have been reported (Cliffton, 1949; Kernan, 1950; Clain and Heffernan, 1956).

I should like to thank Mr. G. F. Langley for allowing me to publish this case, and for his helpful criticism in the preparation of the paper.

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DUODENAL DIVERTICULUM CONTAINING A STONE, COMPLICATED BY PERFORATION

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FROM a clinical point of view duodenal diverticula are of three types, two very common and the third uncommon.

Diverticula of the first part of the duodenum due to peptic ulceration are formed either by epithelialization of a large ulcer crater, or by pouching of the wall opposite an ulcer which has shortened its own side by scarring. These diverticula are frequently found on X-ray examination and at operation.

Diverticula of the second, third, and fourth parts of the duodenum are common radiological and post-mortem findings and are probably present in between 2 and 5 per cent of patients over 50 years of age. They

FIG. 750.—Stone from perforated duodenal diverticulum (scale in centimetres).

are mostly acquired protrusions of mucosa and submucosa through the muscular coat. Congenital pouches of all the layers of the duodenal wall would seem to be very much less common.

Very occasionally diverticula may be formed in the duodenum as a result of disease of the gall-bladder. These are either traction diverticula resulting from the fibrous shrinkage of an adherent gall-bladder or are due to the persistence of part of a cholecysto-duodenal fistula as a result of retained gall-stones (Harley, 1857; Hunt and Herbst, 1925).

In general the term 'duodenal diverticulum' is taken to refer to the herniations so common as an incidental finding on barium-meal examination. About two-thirds of them are on the concave aspect of the second part of the duodenum near the ampulla of Vater, where duodenal branches of the pancreatico-duodenal arteries may be associated with points of decreased resistance to herniation through the duodenal wall. Probably less than 10 per cent are multiple (Weintraub and Tuggle, 1941).

Duodenal diverticula rarely give rise to symptoms requiring surgery, but a wide variety of complications have been recorded (Ogilvie, 1941), two of the rarest being perforation and the presence of stone. This paper is the first record of a case of perforation of a duodenal diverticulum with the extrusion of an enterolith.

CASE HISTORY

M. P., a female aged 62 years, was admitted as an emergency on Aug. 17, 1959, having been quite well until 24 hours before admission, at which time a right lateral subcostal pain developed, which was continuous and fairly severe and was accompanied by vomiting. Ten hours before admission pain developed in the right shoulder, and 6 hours before admission the pain moved suddenly to the centre of the abdomen, becoming much more severe. There was no jaundice, no urinary symptoms,

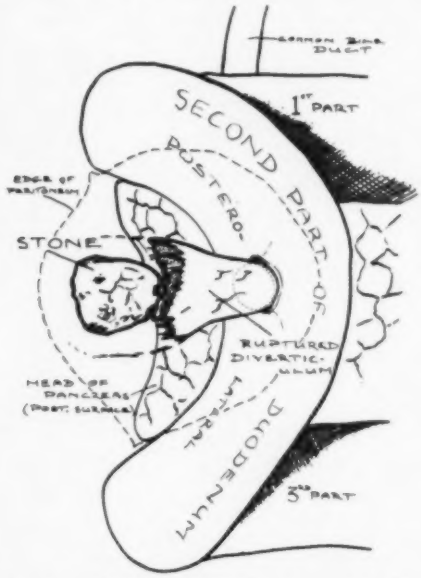


FIG. 751.—Diagram of the findings at operation. The second part of the duodenum has been rotated forwards and to the left so that its posterior wall faces almost to the front. The dotted line represents the edge of the peritoneum, which has been separated along the lateral border of the second part of the duodenum. Thus the area enclosed by this dotted line in the diagram is entirely retroperitoneal.

and the bowels were open normally. On examination her temperature was 100·4°, her pulse 84, and her blood-pressure 150/90. Her tongue was rather dry. There was abdominal tenderness maximal in the right subcostal region, with well-marked rebound tenderness. Liver dullness was unimpaired and bowel-sounds were scanty but normal. Plain radiographs showed no free gas in the peritoneal cavity, no evidence of obstruction, and no opaque biliary calculi.

The diagnosis made was acute cholecystitis, the differential diagnosis being subhepatic appendicitis. Operation was advised and preparation for this included intravenous drip and gastric suction.

AT OPERATION (R. V. F.).—Under general anæsthesia the abdomen was explored through a right paramedian incision. There was a small quantity of light-brown, clear fluid in the peritoneal cavity but no other evidence of peritonitis and no free gas. The gall-bladder was tensely filled but normal in size and appearance, and no stones were palpable in it or in the cystic duct. The common bile-duct was normal in appearance and size and on palpation no stones were detected. The appendix was not inflamed. There was localized scarring in the prepyloric area of the stomach, where a tag of omentum was adherent. The second part of the duodenum and the head of the pancreas were pushed forward by gross œdema of the retroperitoneal tissues and a little yellow fluid was visible behind the peritoneum above the first part of the duodenum. The pancreas itself felt thicker than usual, but was of normal colour and was not injected. The peritoneal reflection on to the posterior abdominal wall from the lateral side of the second part of the duodenum was attenuated and the finger of the left hand could be passed behind this part of the duodenum from the lateral side without formally dividing the peritoneum. A hard lump was felt behind the head of the pancreas, which proved to be a stone (*Fig.* 750), about 3 cm. in diameter, which had ulcerated out of a retroperitoneal duodenal diverticulum arising from the posterior aspect of the second part of the duodenum slightly proximal to the level of the ampulla of Vater. The stone was lying free in œdematous retropancreatic areolar tissue and the fundus of the diverticulum had completely disintegrated, leaving only a cuff of the sac lined by mucosa and with a ragged, black, necrotic, free distal border. The orifice of the diverticulum was brought into view by rotating the second part of the duodenum forwards and to the left, and the continuity of the mucosa of the remains of the diverticulum with that of the duodenum was demonstrated. The orifice was unobstructed and admitted the tip of the index finger (*Fig.* 751). The stone was removed, the common bile-duct explored by a longitudinal incision in its supraduodenal part and, no stone being found, a T-tube was inserted with its long lower arm passing through the ampulla into the duodenum. To obtain a better view of this part of the operation and to avoid damage to the ampulla or common bile-duct, the orifice of the diverticulum was extended by inserting one blade of a pair of scissors through the orifice into the duodenum and cutting the posterior duodenal wall obliquely downwards and laterally (*Fig.* 752). The remainder of the diverticulum was excised and the duodenal wall was closed with two layers of continuous catgut sutures, care being taken to avoid injury to the ampulla of Vater and the common bile-duct. A corrugated rubber drain was passed through a stab incision in the right flank and the T-tube was brought out from the top of the main abdominal incision, which was closed in layers.

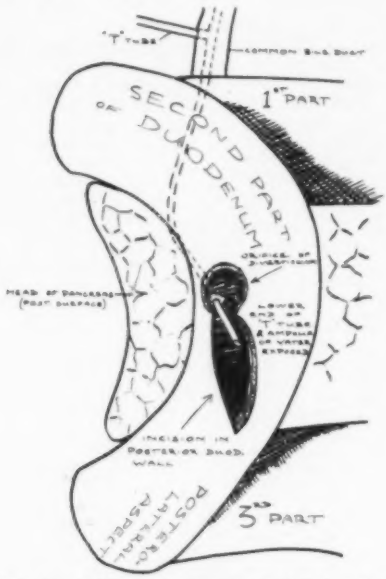


FIG. 752.—Diagram to show the duodenotomy incision made from the orifice of the diverticulum which has been excised. The T-tube is in place in the common bile-duct.

Post-operatively the patient received a course of penicillin and streptomycin by injection. She made an excellent recovery apart from a wound infection and some transient urinary retention. Cholangiography on the ninth post-operative day outlined a normal biliary system and the T-tube was therefore removed. Subsequent healing was uneventful and the patient was discharged to a convalescent home on Sept. 8.

The stone had a hard irregular surface, but the interior was composed of homogeneous finely granular bile-stained material. Chemical examination showed that this was not a gall-stone but was an enterolith with scarcely any inorganic matter in it and no cholesterol.

DISCUSSION

1. Perforation of a Duodenal Diverticulum.—The 11 cases found in the literature, together with the case now reported, are summarized in *Table I.* The case recorded by Huddy (1923) is included because the tip of the diverticulum was black and gangrenous, perforation was imminent, and in clinical features and immediate operative findings the case closely resembled that now reported. Lucinian (1930) described a case as a perforated diverticulum which was more in the nature of an ulcer in a diverticulum penetrating the pancreas. It has not been included.

In the 12 cases of perforation the sexes were equally represented, the age range being from 27 to 80 years. In the 10 cases in which details are given the diverticulum and the perforation were retroperitoneal and in 9 of them it was arising in the second part, mostly medially and peri-ampullary, but that of Glasier and Corbett (1952) laterally. Even in this case the duodenal contents and resulting cellulitis were confined to the retroperitoneal spaces, and this seems to be the general rule. Several reports mentioned the presence of a small quantity of yellow or brown fluid in the peritoneal cavity without frank generalized peritonitis. This fluid possibly results from the inflammation of the peritoneum overlying the area of cellulitis and is probably not duodenal contents which have escaped from the retroperitoneal space. The perforation was almost invariably at the apex of the diverticulum, possibly because that is the point farthest from the blood-supply. There is no evidence that obstruction of the neck of the diverticulum is a factor in the aetiology of perforation. No case other than that now presented reported a stone in the perforated diverticulum, but in 2 cases (Monsarrat, 1926; and Jacob and Lister, 1958) the sac contained yellow or orange paste-like material, and in Huddy's (1923) case there was some friable golden-yellow debris present.

The pre-operative diagnosis was perforated duodenal ulcer in 4 cases, acute cholecystitis in 3

cases, acute appendicitis in 2 cases, and high intestinal obstruction in 1 case. Mousseau and de Ferron (1956) diagnosed a perforated abdominal viscus. Alternative diagnoses included acute pancreatitis, perforated diverticulitis coli, and mesenteric occlusion. The clinical picture is like that of a perforated peptic ulcer in the severity of the pain and the rapidity of its onset and progression, more like that of an acute cholecystitis in the localization of the pain and tenderness and in the pyrexia and vomiting, and more like an acute appendicitis in the degree of tenderness, guarding, and rebound tenderness found on examination.

Table I.—PERFORATION OF DUODENAL DIVERTICULUM

AUTHOR	YEAR	SEX	AGE	PRE-OPERATIVE DIAGNOSIS	OPERATION	DIVERTICULUM	PERFORATION	RESULT: RECOVERED OR DIED	REMARKS
Huddy	1923	F.	27	Acute subhepatic appendicitis	Excision of diverticulum	Peri-ampullary	Apex black and gangrenous	R	Perforation was imminent
Monsarrat	1926	F.	58	Acute cholecystitis	Excision of diverticulum	Peri-ampullary	Apical	R	Diverticulum full of paste-like material
Levering	1933	M.	80	Perforated peptic ulcer	Perforation of D.U. repaired	?	Base of diverticulum	D	Coincidental perforation of diverticulum found at autopsy
Beaver	1938	M.	54	1. Acute appendicitis 2. Perforated D.U. 3. Perforated diverticulitis	Perforation not found	Peri-ampullary	Apical	D	Perforation found at autopsy
Boland	1939	M.	59	Perforated peptic ulcer	Perforation not found	Peri-ampullary	Apical	D	Perforation found at autopsy
Byerly	1949	M.	71	Perforated peptic ulcer	Excision of diverticulum	Peri-ampullary	Apical	R	
Glasier and Corbett	1952	M.	67	Not stated	Perforation not found	Second part of duodenum, lateral	Apical	D	Perforation found at autopsy
Ferguson	1953	F.	58	1. Perforated P.U. 2. Acute pancreatitis 3. Mesenteric occlusion	Diverticulum excised	Peri-ampullary	Apical	R	
Mackay and Page	1955	F.	62	High intestinal obstruction	Diverticulum excised	Third part of duodenum	? Apical	R	
Mousseau and de Ferron	1956	M.	67	Perforated viscus	Perforation not found	?	?	R	
Jacob and de Lister	1958	F.	67	Acute cholecystitis	Diverticulum excised	Peri-ampullary	?	R	A fistula developed and this was successfully repaired at a second operation
Fiddian	1959	F.	62	Acute cholecystitis	Diverticulum excised and stone removed	Peri-ampullary, containing a stone	?	R	Stone extruded from the diverticulum. Duodenum and common bile-duct explored at operation

At operation there is no free gas in the peritoneal cavity, but there is usually some yellow or brown fluid. There is œdema and swelling of the retroperitoneal tissues in the region of the duodenum, and more fluid, which may be bloody or bile-stained, is often visible through the peritoneum.

Retroperitoneal exploration of the duodenum is usually needed to find the perforated diverticulum which should be excised. The neck of the sac is in most cases close to the ampulla of Vater and it can be argued that duodenotomy or choledochotomy is advisable to avoid damage to the common bile-duct or ampulla during closure. Byerly (1949), on the other hand, to minimize this risk, excised only four-fifths of the diverticulum, closing and plicating the remainder. Glasier and Corbett (1952) advise drainage only, to be followed by excision and closure at a second operation. There is only 1 case on record (Mousseau and de Ferron, 1956) to give any support to this suggestion.

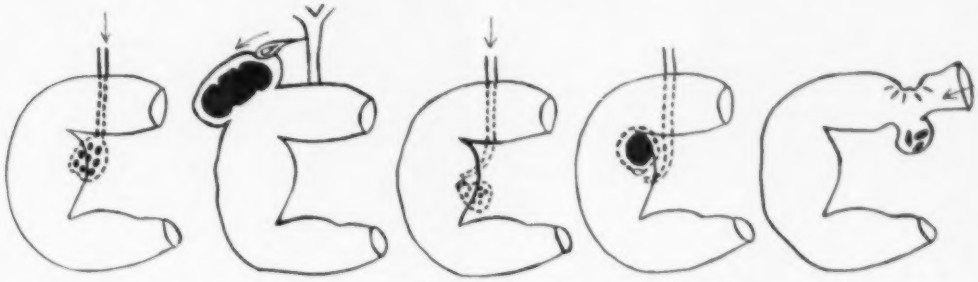
In 7 of the 12 cases the perforated diverticulum was found and excised, and these 7 patients all survived. Of the 5 cases in which the perforation was not found at operation only 1 survived. This was the case of Mousseau and de Ferron (1956) in which a fistula developed and persisted until successfully closed at a second operation.

Cattell and Mudge (1952) state that surgical treatment of duodenal diverticula for non-acute complications is accompanied by a significant mortality, and among the complications of such treatment are leakage at the suture line with peritonitis and pancreatitis, pancreatic fistula, and injury to the common bile-duct. None of these was a serious problem in this series, although 1 case (Jacob

and Lister, 1958) reported bile-stained discharge for three weeks post-operatively.

2. Stone in a Duodenal Diverticulum.—Cases of duodenal diverticulum complicated by the presence of a stone or stones seem to be less rare than cases of perforation. *Fig.* 753 illustrates some of those reported. In addition there have been several cases of intestinal obstruction due to the impaction of an enterolith which was thought to have arisen in a duodenal diverticulum (Hellstrom, 1936; Shaw, 1940; Goodall, 1948; Slater, 1953; and Atwell and Pollock, 1960). Armitage, Fowweather, and Johnstone (1950) described a similar case in which a second stone was present in a duodenal diverticulum, confirming the site of origin of the obstructing stone. In reported cases females predominate over males in a ratio of 8 to 1, and ages ranged from 46 to 80 years.

A confirmed pre-operative diagnosis of stone in a duodenal diverticulum has never been recorded. Duodenal enteroliths are for the most part of insufficient radiopacity to throw a significant shadow on a plain radiograph of the abdomen, but the case reported by Rankin (1942) is an exception. In his patient a large radiopaque stone was seen, but it was thought to be in the gall-bladder until operation showed it to be in a duodenal diverticulum. Armitage and others (1950), Glasier and Corbett (1952), and Atwell and Pollock (1960) all recorded the demonstration of a filling defect in a duodenal diverticulum but diagnosis of stone was not made before operation.



CHOMEL (1710)
Gall-stones in a dilated lower end of common bile-duct.

HARLEY (1857)
Gall-stones in a diverticulum resulting from inflammatory gall-bladder disease.

HARE AND CATTELL (1944)
Gall-stones in a primary duodenal diverticulum.

OGILVIE (1941) (and author's case)
Enterolith formed in a primary duodenal diverticulum.

LEVY (1956)
Apricot stones in an 'ulcer' diverticulum of the first part of the duodenum.

FIG. 753.—Stones in duodenal diverticula: different entities (very diagrammatic). The arrows indicate the path of the stones.

True enteroliths forming in duodenal diverticula are usually solitary, but may be multiple (Ward-McQuaid and Gordon, 1950; Armitage and others, 1950). They are said to consist largely of choleic acid (Armitage and others, 1950; Atwell and Pollock, 1960), and a portion of fruit skin or a fruit stone has often been found at their centre. Stasis resulting from the absence, usually, of muscle from the wall of the diverticulum must be a significant aetiological factor, although in the case of Ward-McQuaid and Gordon (1950) all layers of the duodenal wall were present in the sac.

Symptoms and complications are varied. In some cases 'biliary' symptoms were attributed to the presence of stones (Harris, 1932; Rankin, 1942; Glasier and Corbett, 1952). The pylorus was obstructed in the case of Hunt and Herbst (1925), and Ogilvie's (1941) patient died from acute pancreatitis to which he considered the stone had largely contributed by pressure on the head of the pancreas. The most serious risk is intestinal obstruction from impaction of the stone lower down the bowel, and it may be well to remember that a stone might appear too small to cause a mechanical obstruction yet will do so by initiating spasm in the bowel wall and, probably because of this fact, the obstruction may be intermittent (Fiddian, 1959). This paper is the first record of perforation complicating the presence of a stone in a duodenal diverticulum.

Glassman (1929) described a large diverticulum of the duodenum in which a concretion had lodged for two years and which was expelled during a routine radiographic examination. However, his evidence is circumstantial and not convincing.

Treatment will usually be undertaken for some complication resulting from the presence of stone(s) in the duodenal diverticulum, and the details of management will be dictated by the nature of the complication. In the case reported by Hunt and Herbst (1925) the diverticulum, full of gall-stones, surrounded and obstructed the first part of the duodenum. There was a fistulous connexion with a chronically inflamed gall-bladder. A good result was obtained by partial gastrectomy and cholecystectomy. The treatment of perforation of a duodenal diverticulum has already been discussed. Hæmorrhage is a serious complication of duodenal diverticula, which might be thought more likely to occur and to require operation in the presence of stone. However, such a combination has not been reported. Intestinal obstruction by an enterolith extruded from a duodenal diverticulum will usually be treated by enterolithotomy, but Atwell and Pollock (1960) advise crushing the enterolith in the bowel. This may be attempted if the presence of a normal gall-bladder excludes gall-stone ileus, which should be treated by enterotomy (Fiddian, 1959). It is probably unnecessary to deal surgically with the diverticulum in such a case of obstruction unless another stone or stones are contained in it (Armitage and others, 1950), in which event the stones may be removed by duodenotomy or diverticulectomy.

Of 8 cases in which a direct surgical attack was made on a duodenal diverticulum containing a stone or stones, 2 died (Rankin, 1942; Atwell and Pollock, 1960) in the post-operative period, the remainder making satisfactory recoveries.

SUMMARY

A case of perforation of a duodenal diverticulum with extrusion of an enterolith is reported. The literature on duodenal diverticula complicated by the presence of stone or by perforation is reviewed. It is concluded that the treatment of perforation is diverticulectomy and that the treatment of stone is that of its complications. This paper is the first report of perforation complicating the presence of a stone in a duodenal diverticulum.

Acknowledgement.—I wish to thank Mr. John Hosford for permission to publish this case and for his helpful advice and criticism.

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SPONTANEOUS RUPTURE OF THE STOMACH

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SPONTANEOUS rupture of the stomach is a very uncommon condition and such cases as have been reported have usually died. In 1952 Rejthar was able to find only 1 previous survival in the British literature to add to his report. This paper presents a case which is unusual not only because the patient survived the event, but also because radiographs taken before operation show features which are of value in establishing the diagnosis.

CASE REPORT

Mrs. E. S., a housewife aged 49 years, was admitted to hospital in October, 1959, complaining of abdominal pain of 4 hours' duration. For ten years she had experienced periodic bouts of epigastric pain after food, relieved by alkali and sometimes accompanied by vomiting. It was her habit to belch large quantities of wind after a meal.

On the day of admission she had been knitting in a chair two hours after her lunch, when she developed upper abdominal pain which rapidly increased in severity. No vomiting occurred.

ON ADMISSION.—The patient was obviously in pain and had cold cyanosed extremities. B.P. 150/90, pulse-rate 100/min. T. 98° C. The abdomen was extremely distended, tender in its upper half, and tympanitic on percussion. Rigidity, whilst generalized in extent, was much less in degree than that accompanying the early stages of a perforated peptic ulcer. Bowel-sounds were not audible.

A clinical diagnosis of volvulus, probably of the cæcum, was made and, after plain radiographs of the abdomen were taken, a gastric tube was passed.

AT OPERATION.—Under general anæsthesia the abdomen was found to be of normal contour and quite without the previously remarkable distension. A right paramedian incision was made. The peritoneal cavity contained a large quantity of gas but no obvious fluid or food particles. Chronic anterior and posterior duodenal ulcer scars were present, but perforation of these had not occurred. The stomach was large in size and on the lesser curvature, high up, midway between the anterior and posterior surfaces, was a 2-in.-long vertical split in the serosa and muscle. On spreading this split open laterally, a hole in the mucosa was revealed, ½ in. across. The gastric wall about the split was free of induration or œdema and subsequent histological examination of an operative biopsy taken from this site revealed no evidence of peptic ulceration. The rupture was repaired in two layers with catgut and the abdomen closed.

The post-operative course was uneventful and to date, six months since the operation, the patient has remained free of symptoms so that no further surgery has been undertaken for her chronic duodenal ulceration, presumably now quiescent.

DISCUSSION

Any healthy muscular organ possessing sphincters at inlet and outlet is liable, albeit rarely, to spontaneous rupture, and Cronin (1959) has reviewed such an event occurring at different sites in the

alimentary canal. The diagnostic criteria in the case of gastric rupture have been suggested by Millar, Bruce, and Paterson (1957) as consisting of: (1) Severe abdominal pain; (2) Tympanitic distension; (3) Abdominal rigidity; (4) Subcutaneous emphysema; and (5) Evidence of shock.

In the case reported here all these features with the exception of subcutaneous emphysema were present to a varying degree, and it is the detection of such emphysema, above all else, which would lead one to the correct diagnosis on clinical grounds. The abdominal distension is due partly to the presence of a pneumoperitoneum and partly to the gaseous distension of the stomach. When the air in the peritoneal cavity is under tension, as in the cases reported by Millar and others (1957), subcutaneous emphysema may be expected to develop, but when the pneumoperitoneum is small compared to the degree of gastric distension, as in the case here, emphysema would probably not develop. The same diagnostic significance of subcutaneous emphysema occurs in cases of spontaneous rupture of the œsophagus, where the patient with severe pleural and also abdominal pain, dyspnœa, and shock, shows emphysema in the tissues of the suprasternal and clavicular fossæ. Since spontaneous rupture of both the œsophagus and stomach are frequently diagnosed as cases of perforated peptic ulcer, it behoves the surgeon to examine deliberately for evidence of subcutaneous emphysema in every such instance.

When the radiographs obtained in this patient were considered again after operation, they were seen to have the following features of importance. *Fig.* 754 is a plain radiograph of the abdomen. It shows an enormously distended stomach and a small pneumoperitoneum. Close inspection of the outline of the lesser curvature (*Fig.* 755) reveals a break in the continuity which anatomically would correspond with site of the rupture as seen at operation. The arrow in each figure points to the site.

It is probable that the sequence of events in this patient was that, as a result of aerophagy and perhaps pylorospasm due to her chronic duodenal ulceration, the stomach became distended to the point of rupture. Having ruptured and leaked some air, the rent in the gastric wall then held, so that the radiographs taken at this stage revealed the features shown in *Figs.* 754, 755. Subsequently, the passage of a gastric tube so deflated the stomach that her abdomen was of more normal contour by the time of operation.

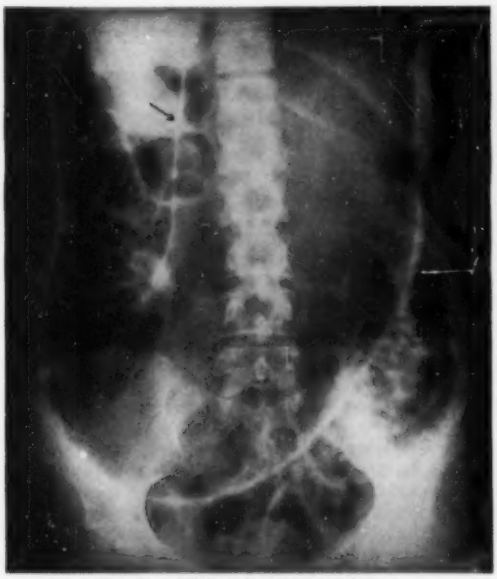

FIG. 754.—Plain radiograph of abdomen.

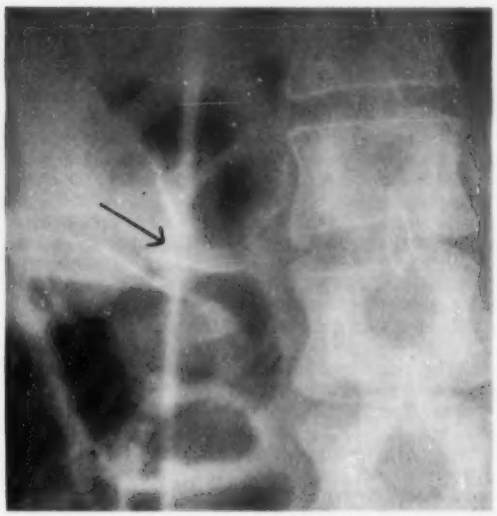

FIG. 755.—Detailed view of site of rupture.

SUMMARY

1. A case of spontaneous rupture of the stomach with survival is presented.

2. The importance of examining for subcutaneous emphysema in patients upon whom a diagnosis of perforated peptic ulcer has been made is stressed.

3. The radiographs taken in this case are also reported for their diagnostic value.

I wish to thank Professor R. Milnes Walker and Mr. J. A. Pocock for permission to publish this report and for their advice.

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PROBLEMS OF ACHALASIA

By DONALD BARLOW, London

THIS paper is based on an experience of 61 cases of achalasia seen between the years 1936 and 1960.

True reflex spasm of the cardia is not under consideration. It is cured by the removal of the exciting cause.

In 1876 Zenker studied 17 cases of "simple ecstasy of the œsophagus" and in 1884 von Mickulicz (1904) coined the name "Cardio-spasm".

Between the years 1908 and 1912 Plummer (1912) and his colleagues in the Mayo Clinic collected a large series of cases and studied methods of diagnosis and treatment. From this work came the Plummer's bag dilator, later modified by Negus in this country. This bag gave great relief to many sufferers, but, because of the risks involved and the fact that it did not really take into account the pathology involved, was later largely superseded.

In 1903 G. Marwedel devised a method of approach to the cardiac end of the stomach by making an incision parallel with the left costal margin and dividing costal cartilages with ribs 7, 8, and 9 and hinging up the whole left costal margin.

E. Heller in 1914 used this approach in his now classical operation of œsophagoplasty. He believed that the cause of the lesion was a true spasm of the circular muscle of the lower 3 cm. of the œsophagus, but he recorded that there was no hypertrophy of muscle in the affected segment. He was the first to see, however, that there was gross and obvious hypertrophy of the œsophageal musculature above the segment. He noted, too, that even under deep anæsthesia the contracted cardiac segment did not relax, and he therefore applied the principle of Rammstedt and divided the longitudinal and circular muscle fibres over the whole affected area. He did this both on the anterior and posterior surfaces in order to ensure an adequate bulge of the mucosa and to effect complete relief. He made no comment in his paper about the obvious contrast between the grossly hypertrophic muscle lesion of the pyloric sphincter and the thin attenuated type of muscle at the lower end of the œsophagus with the hypertrophy confined to the area above it.

The principle of Heller's operation is still considered the most effective method of relief to-day, but the operation itself was simplified to its present form in 1942 by Barlow, who used an ordinary paramedian incision and a single anterior division of the musculature.

In 1924 Vinson presented a detailed review of 415 patients. He noted that many cases were clearly of long standing before symptoms were sufficiently marked for patients to seek advice. The muscular hypertrophy above the lesion had kept pace, as it were, for long periods with the increasing obstruction. In the long run there was 'œsophageal decompensation' often corresponding with the first symptoms of illness. By this time there was sometimes a mega-dolicho-œsophagus, perhaps clearly visible as a grossly widened mediastinum in a plain chest radiograph. Œsophageal peristalsis was flagging with the 'upstream mega'.

PATHOGENESIS

The paper by Deloyers, Cordier, and Duprez, published in 1957, on experimental creation of cardiospasm in the cat also reviews clearly the historical background of the disease and the evidence supporting the view that the trouble is due to localized degeneration of Auerbach's plexus.

The cause of the degeneration is still unknown and is therefore not yet preventable. There is no established evidence that it is congenital.

Chevalier Jackson's term 'phrenospasm', indicating responsibility of the diaphragm, has long since been discounted.

Let us list the evidence with reference to pathology:—

1. The ages of patients seeking advice in the writer's series of 61 cases varies from 6 weeks to 72 years.

The baby's lesion was classical and completely relieved by the Heller operation, but is unique in the series. The youngest patient apart from this was 14 years, but 7 were 25 years of age or younger.

2. There was never any hypertrophy of the obstructing segment—only hypertrophy of both circular and longitudinal muscle above it. This lack of hypertrophy in the segment indicates lack of work done and confirms Hurst's observation of 1913 that, since a mercury bougie could be swallowed so easily, spasm could not be the cause. That a full-size œsophagoscope can be passed on œsophagoscopy also supports this. Further research and histological studies of all parts of the œsophagus are indicated.

3. Severity of symptoms is not always commensurate with the degree of œsophageal dilatation and lengthening.

4. Screening radiography of the œsophagus using opaque media shows active peristalsis above the cardia, but the wave fails to pass the latter except that at varying intervals a small squirt will go through. There is always a residue above. There is clearly either an absence of relaxation of the circular fibres or a segment through which peristaltic waves will not pass.

5. The gradual onset of the disease suggests a degenerative neurological process. It is believed that Keith (*c.* 1903) showed that the plexus at the lower end of the œsophagus contained a large admixture of nodal tissue similar to that present in the A–V bundle of the heart.

6. At a late stage of the disease, secondary œsophagitis may develop, but this is never seen early and there is no indication on œsophagoscopy using a high-power telescope that any inflammation precedes the onset of the lesion. Rake, in 1926, found around the plexus of Auerbach an infiltration of lymphocytes and eosinophils. In view of Keith's work he thought the changes in Auerbach's plexus comparable to those occurring in Stokes-Adams disease. Hurst (1924) had already foreseen that achalasia was caused by progressive organic disease involving Auerbach's plexus. This finding has been repeatedly confirmed since by Cameron in 1927,

Mosher and McGregor in 1928, Beattie in 1931, and by Lanzara in 1949. Rosenheim in 1900 raised the question of possible disease of pneumogastric (vagal) nerves, but no degeneration of these nerves has been demonstrated.

Sir Cooper Perry proposed to call the complaint 'achalasia'.

7. Auerbach's plexus is found with its myenteric ganglia between the circular and longitudinal smooth-muscle layers. It extends from œsophagus to rectum. It is parasympathetic and separate from the sympathetic fibres which pass directly to the muscles. There is no demonstrable connexion between Auerbach's ganglionated parasympathetic plexus and the sympathetic fibres within the gut. The degenerative process which has been demonstrated in achalasia leaves the sympathetic fibres intact and functional. Hence, as pointed out by Lendrum in 1937, destruction of the parasympathetic will tend to leave sympathetic predominance. At sphincters this theoretically results in contraction or failure of relaxation of the circular fibres, and at non-sphincter levels in atonia of them. Although the cardia is not anatomically a sphincter in the ordinary sense, there would appear to be sphincter action there in this sense. Achalasia seems to be due to failure of relaxation of the circular muscle-fibres of the lower segment of the œsophagus.

Wangensteen (1951) noted this contractile asynchronism and called it 'dystonia'.

8. Lesions similar to achalasia in which there is degeneration of Auerbach's plexus with partial or complete disappearance of the myenteric ganglia are now well authenticated and these are not confined to sphincter regions.

Robertson and Kernohan (1938) showed that such degeneration occurred in Hirschsprung's disease. In 1949 Etzel, Recalde, Moore, and Netto are said to have found 8 cases of combined cardiospasm and Hirschsprung's disease and noted the atrophied Auerbach nerve-cells in both sites. The author has been unable to find their paper.

Arrigo Raia in 1955 reported, in addition to 69 cases of mega-œsophagus, 7 cases of achalasia of the pylorus and 2 cases of megajejunum and mega-ileum, whilst megaduodenum with Auerbach degeneration was reported in 1955 by Barnet and Wall. The author has seen a case of this in which there was gross dilatation of the whole duodenum as far as the beginning of the jejunum but no narrow area as in achalasia. Gastrojejunostomy resulted in complete relief in this case.

Unfortunately, histological examination was not carried out but the appearance macroscopically indicated a short segment near the duodenojejunal junction distal to the dilated and hypertrophied duodenum through which peristalsis waves failed to pass. It was thought at the time that the segment, some 3 cm. long, was denervated.

From the above, it would appear inadvisable to consider achalasia in isolation. Deloyers and others (1957) found difficulty in explaining the obstruction in achalasia, Hirschsprung's disease, and allied sphincteric and non-sphincteric lesions on the basis of residual sympathetic preponderance, but they agreed that Auerbach degeneration or disappearance was the common factor in all. Their difficulty was that, theoretically at least, the circular muscle in the affected segment at sphincter level is either permanently contracted or fails to relax whereas, at non-sphincter level, it is, or should be, in a state of atonia.

The fact is that in both cases there is an obstructing segment and peristaltic waves will not pass through it. It seems to the writer that an atonic segment can and does act as just as great an obstruction as if it had been completely denervated.

It now remains to determine what are the factors which produce the Auerbach plexus degeneration. No information upon this question has so far come to light.

SYMPTOMS AND DIAGNOSIS

Where a flask-shaped dilated œsophagus is seen in a barium swallow, there is a tendency to accept the view, without further investigation, that the diagnosis is achalasia. Nothing is more dangerous. Careful investigation, in particular with the use of a high-power telescope through an œsophagoscope, is essential.

Cases of achalasia present in a variety of ways:—

1. No symptoms. Œsophageal dilatation discovered on plain X-ray examination of the chest or upon barium swallow.

2. Increasing dysphagia. Sense of fullness behind sternum. Regurgitation of food is much less common than might be expected, but patients will sometimes make themselves sick by putting the fingers down the throat. Sometimes they complain of choking. There are cases with gross œsophageal enlargement with very little in the way of symptoms.

3. Increasing weakness and wasting and anæmia. Rarely scurvy or beri-beri.

4. Chest troubles. Cough, dyspnœa, purulent sputum, pain in the chest, fever, asthma. It is recognized that where a chest radiograph reveals patchy pneumonitis, especially where there is no gastric air bubble, chronic œsophageal obstruction may be safely diagnosed. Absence of gastric air bubble is diagnostic of œsophageal obstruction. The chest lesions which may result in lung abscesses or bronchiectasis occur from œsophageal 'spill-over' and indicate the need for investigation of that organ.

5. On rare occasions, brain abscess from the respiratory lesions secondary to achalasia has been recorded.

Differential Diagnosis.—Differential diagnosis is particularly from hiatus hernia with stricture and from strictures from other causes including carcinoma of the œsophagus or cardia of the stomach. Sometimes pyloric stenosis due to chronic duodenal ulcer may be associated with gross œsophageal dilatation, and in this case there may or may not be a hiatus hernia. If there is, there may also be œsophago-gastric ulceration and stricture. It is salutary to recall cases where carcinoma at the cardia has closely simulated achalasia in X-ray appearance. Biopsy under high-power magnification should not be neglected in spite of the fact that it may be possible to pass the œsophagoscope through into the stomach.

It is clearly necessary to determine by direct inspection the exact level, in relation to the diaphragm, of commencement of gastric mucosa. A gastric-lined œsophagus may confuse the issue. In all instances where achalasia is suspected, careful X-ray screening

is required with facilities for tipping the patient into the full Trendelenburg position. Only thus can hiatus hernia or incompetence be excluded.

Upon a correct initial diagnosis will depend the proper planning of successful surgery.

COMPLICATIONS

These may be listed as follows:—

1. In association with the disease *per se*.
2. In association with investigation and operation.
3. Late operative complications.

1. In Association with the Disease per se.—In this series, by far the most important group of complications concern the chest. 'Spill-over' pneumonitis is seen in other forms of chronic œsophageal obstruction and notably occurs in those who lie flat in bed at night or stoop at their work. The œsophageal contents flow quietly into the respiratory passages.

Cough, particularly in the morning, occurs early. Pneumonitis characteristically affects, alternately, different areas of the lung. The patient learns to live with his 'bronchitis', cough, or asthma. Every now and again he develops bronchopneumonia of variable extent, site, and severity, and slowly goes downhill from wasting, toxicity, and pulmonary insufficiency unless the cause is diagnosed. Fulminating cases with necrosis and hæmoptysis are not common, but 2 cases of multiple lung abscess occurred in the writer's series.

Œsophagitis does not develop so long as the compensatory hypertrophy can cope with the obstruction at the cardia. When decompensation occurs, rapid and progressive dilatation follows and residual food decomposes. Œsophagitis may be slight or severe, acute or chronic. Occasionally it may be very acute and cause pain and even severe hæmorrhage. The latter means ulceration and may be so severe as to result in death. Œsophagoscopy in such cases shows marked œdema with multiple indolent erosions. Such hæmorrhage is difficult to deal with, but can usually be controlled by blood replacement and feeding via a Ryle's tube. An emergency Heller operation is strongly contra-indicated because at this stage the œsophageal mucosa may not be intact and perforation is almost inevitable.

Chronic œsophagitis is more common, but is usually not of such a severity as to contra-indicate fairly early surgery. The obstructive lesion and persistent slight loss of blood from ulceration tends to result in chronic iron-deficiency anæmia and mental depression. The 'eating recluse' is a pathetic figure.

Inanition or starvation, with concomitant scurvy, beri-beri, or other evidence of vitamin deficiency, is more common than might be suspected but is usually not severe. Electrolyte imbalance may rapidly occur, especially where food has suddenly become impacted. It may be necessary to pass a Ryle's feeding tube for 7 to 10 days before operation.

Rarely, when hard food is impacted, spontaneous œsophageal perforation may occur. The mediastinitis which follows this may be fatal or very severe, owing to the gross infection. Empyema from this cause is uncommon.

Stricture of the œsophagus apart from the results of surgery was unknown in the writer's series, but carcinoma was found in the lower end of the dilated œsophagus in 2 of the 61 cases. Both cases had classical achalasia and in neither case was the growth causing any obstruction. It was diagnosed on radiography and confirmed by biopsy.

Simple strictures followed in 6 of the early cases operated upon from below by the modified Heller method because of post-operative reflux and ulceration. Since those days careful attention at operation is given to tightening the cardiac opening of the diaphragm and suturing the lower end of the œsophagus below the diaphragm.

2. In Association with Investigation and Operation.—The œsophagoscope is a dangerous and even lethal weapon in hands untrained in its use. An excellent magnified view is obtainable by using a high-power distally illuminated telescope. A film on the art of œsophagoscopy is in course of preparation.

Apart from the damage to teeth and gums, œsophageal perforation either in the post-cricoid region or towards the lower end is the chief hazard. The post-cricoid region is best negotiated under relaxant anæsthesia with the head held in full flexion and the cricoid lifted forwards on the end of the instrument. Perforation at the lower end is avoided by using the telescope and by remembering that, especially in advanced cases, œsophageal sagging is common. The cardiac orifice is usually much more to the left and anterior than is sometimes realized.

Instrumental perforation may result in severe mediastinitis or empyema, not to mention medico-legal difficulties.

Plummer or Negus bag dilatation and bougies of various kinds, unless used with skill and circumspection, are all potentially dangerous. There is no justification for using any of them unless under the direct guidance of a good illuminated telescope. The writer has seen a severe stricture of the post-cricoid region following trauma in a case of achalasia.

The hazards of Heller œsophagoplasty are not great if care is taken. In the writer's experience the thoracic approach is unnecessarily complicated. Whether the approach is from above or below is probably of little moment, but to do an effective muscular split the diaphragmatic hiatus has to be enlarged sufficiently to draw up or down the fundus so that the split can be extended onto it. In most series the incidence of later hiatal insufficiency is considerable unless measures are taken to prevent it. These are simpler from below.

Perforation of the mucosa has been known to result in empyema or subphrenic abscess.

3. Late Operative Complications.—As time goes on it becomes increasingly apparent that in future greater care will be required to obviate, as far as possible, œsophageal reflux after the Heller operation.

Of the 61 operated cases in the present series, all but 2 had a Heller operation. They were the cases with carcinoma. All but 1 were relieved of their dysphagia and concomitant symptoms.

Later, apart from the 6 quite severe strictures which developed at periods of 1 to 6 years, there were some 15 others with some degree of post-operative reflux and several of them have slight dysphagia.

The rest have for the most part had a recent follow-up and are well and symptom-free. One has a

duodenal ulcer and one a gastric ulcer with a duodenal diverticulum.

The one not at once relieved by operation was an emaciated elderly man with gross œsophagitis and gross sagging of a huge œsophagus. He ultimately died after an œsophago-gastrostomy.

It will be seen that post-operative reflux, with risks of later ulcer, stricture, and chest and other complications, is not uncommon after a straightforward Heller operation. The trouble may not develop until years later. This fact, i.e., that the end-result may be as bad as or worse than the original achalasia, has been realized by the writer for some five years. Investigations revealed three groups of cases:—

1. Those where the cardiac orifice of the diaphragm had been made incompetent with the added risk of incompetence occasioned by the destruction of such natural sphincter action and muscularis-mucosal 'rosebud' control as normally exists. This risk can be obviated in most cases at operation.

2. Those where the vagi were damaged or destroyed, with the added chronic pyloric obstruction which is so liable to follow in such cases. The vagi should not be damaged but, if they are, a pyloroplasty is required.

3. A combination of (1) and (2).

TREATMENT

In spite of the risks involved, it is now generally accepted that the Heller principle is the best available except in extremely advanced cases, because of gross secondary changes as with stricture following ulceration or the appearance of carcinoma. In those events, resection of the lower œsophagus with a Roux-en-Y or a jejunal implant on a vascular pedicle brought up through the mesocolon and lesser omentum and anastomosed above to the œsophagus and below to the fundus of the stomach may be required. The diaphragm can then be well repaired.

Bag dilatation and the use of bougies for achalasia, except as a temporary measure, are superseded by œsophagoplasty.

SUMMARY

If a Heller operation is done, in the writer's view the following precautions are necessary:—

1. It should be done through a good long left paramedian incision up to the costal margin. Long Deaver retractors and good lighting are essential.

2. Great care is required to preserve intact the vagi. If they are inadvertently damaged, a pyloroplasty is required.

3. The fundus must be sutured below the diaphragm and the cardiac orifice carefully repaired by suturing together the limbs of the right crus until it is snug.

4. Three months, 6 months, 1 year, and 5 years after operation, carry out radiography and tip into the Trendelenburg position to exclude reflux. The patient should avoid stooping and lying flat as far as possible.

Now that Auerbach's plexus 'degeneration' (or absence) has been pin-pointed in this and similar lesions as the operative anatomical lesion, it is hoped that the cause of this 'degeneration' will be revealed and become preventable.

It may be, however, that there is a congenital absence or imperfect development of the plexus.

No record has been found of a demonstration of the plexus in stages of degeneration.

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SPLENIC ANEURYSMS

By J. N. WARD-McQUAID, Mansfield

Aneurysm of the splenic artery is a well-recognized condition. Nevertheless recovery after rupture is very rare, and pre-operative diagnosis is uncommon. The main purpose of this paper is to record a patient with severe symptoms cured by removal of the spleen together with the tail of the pancreas, in which was embedded a calcified splenic aneurysm. Four other patients in whom calcified splenic aneurysms were diagnosed as incidental findings are also described briefly. Ten patients with splenic arterial aneurysms have been seen in Mansfield in the last five years. The first five patients have been described elsewhere (Ward-McQuaid, 1958).

Fig. 756.—*Case* 1. Pre-operative radiograph showing calcified splenic aneurysm.

Fig. 757.—*Case* 1. Radiographs of the spleen after removal, showing the aneurysms.

CASE REPORTS

Case 1.—**Relief of severe symptoms after excision of calcified splenic aneurysm.**

P. A., a female aged 63 years, was referred to Dr. R. J. Twort in 1958 as an urgent case. She complained of recurrent attacks of pain in the left upper abdomen, lower ribs, and renal angle for nine months. During the attacks the pain was constant, with exacerbations, and lasted up to two weeks, with similar remissions. There was no radiation, and it was not related to any digestive, bowel, or micturition upsets. It caused her to move about, and was helped by heat. Recently the pain had been severe enough to demand morphine for its relief. She also suffered from belching of wind, constipation, occasional frontal headaches, and breathlessness on exertion. There was no relevant past or family history.

On Examination.—She was a somewhat obese lady, who pointed to the left loin as the site of her pain. There was localized tenderness over the left renal area and lower left ribs posteriorly. Her blood-pressure was 160/90, and there were scattered rhonchi in her chest.

A renal or a pancreatic cause of her symptoms was considered likely.

Investigations.—Blood group O positive; hæmoglobin, white count and differential, fasting blood-sugar, blood-urea, serum-amylase, liver function tests, W.R., urine, and E.C.G. within normal limits. A radiograph of the chest showed moderate cardiac enlargement and probably some congestion in both lungs. A plain film of the urinary tract (*Fig.* 756) showed the calcified shadow of a splenic aneurysm opposite the 12th thoracic vertebra, with smaller opacities laterally, and some calcified mesenteric nodes on the right. No bruit could be heard over the aneurysm.

Diagnosis.—Dr. Twort referred her to me for operation with a diagnosis of splenic aneurysm involving the pancreas.

At Operation.—Under general anæsthesia, given by Dr. H. A. Buck, the abdomen was explored by a left upper paramedian incision. A splenic aneurysm was embedded in the tail of the pancreas. The spleen, most of the splenic artery, the tail of the pancreas, and the associated aneurysm were excised, and also the appendix. There was no other abnormality on laparotomy.

Fig. 758.—*Case* 1. The specimen.

Progress.—Convalescence was uneventful. When seen a year and a half later she had had no more pain since the operation, but still complained of wind.

The Specimen—*Figs.* 757, 758 (now in the Pathological Museum of St. Bartholomew's Hospital, London, No. F.132).—The spleen itself is normal in size. Four aneurysmal sacs are present, the largest of which is 3 × 2½ × 1½ cm. and adherent to a portion of the tail of the pancreas. This aneurysm is at the point of division of the splenic artery, and the smaller aneurysms are on the branches of the splenic artery.

Dr. Stansfeld kindly reported that all these arteries showed a severe degree of atherosclerosis, and that the main aneurysm was also completely filled with old decolorized thrombus. Sections of the aneurysmal wall

show this to be composed almost entirely of old hyaline fibrous tissue, partly calcified. Only a few strands of smooth muscle yet remain. The appearances suggest that this is an atheromatous aneurysm and there is no indication of polyarteritis nodosa or other inflammatory condition. The findings were essentially similar in a section of one of the small aneurysms.

FIG. 759.—*Left*, *Case* 2. Calcified splenic aneurysm: incidental finding in investigation of prostatism. *Right*, *Case* 3. Calcified splenic aneurysm: incidental finding in investigation of melæna.

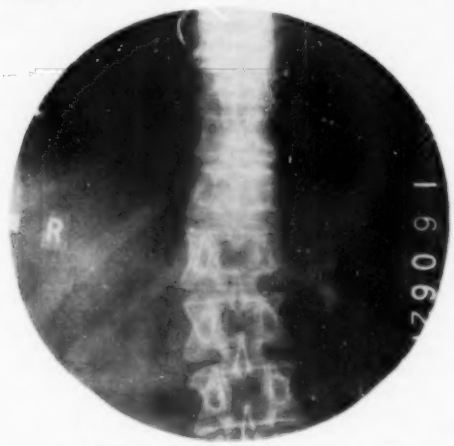

FIG. 760.—*Case* 4. Calcified splenic aneurysm: incidental finding in investigation of dyspepsia.

Case 2.—**Calcified splenic aneurysm: incidental finding with benign prostatic hypertrophy.**

F. C., male aged 71 years. A calcified splenic aneurysm was diagnosed in 1958 at the time of intravenous urography done for prostatism and hæmaturia. It was situated opposite the 10–11th thoracic vertebræ and measured 2 × 1·3 cm. (*Fig.* 759, *left*). The blood-pressure was 145/95. Calcification in the aneurysm was then seen on reviewing the films of an otherwise normal barium enema done in 1954. The patient is well after retropubic prostatectomy, and in 1960 the degree of calcification in the aneurysm appeared unchanged.

Case 3.—**Calcified splenic aneurysm: incidental finding in the investigation of melæna.**

F. G., female aged 66 years. The typical ring shadow of a well-calcified splenic aneurysm was seen in 1958 at the time of an otherwise normal barium meal after recovery from melæna. It was opposite the 11th dorsal

FIG. 761.—*Case* 5. Calcified splenic aneurysm, seen on intravenous pyelography: incidental finding in urological investigation.

vertebra and measured 1·5 × 1·5 cm. (*Fig.* 759, *right*). The blood-pressure was 170/80. Her dyspeptic symptoms had suggested peptic ulceration for many years, and she had had two previous episodes of bleeding. A barium meal done in 1940 is said to have confirmed the diagnosis of duodenal ulcer. The report made no mention of an aneurysm at that time. She is now well and without symptoms.

Case 4.—**Calcified splenic aneurysm: incidental finding in suspected cholecystitis.**

A. E. M., female aged 66 years. Investigation by cholecystography of this obese woman in 1959 revealed a poorly functioning gall-bladder and also the typical ring shadow of a calcified splenic aneurysm. It measured 2 × 2 cm. and was opposite the 11th dorsal vertebra (*Fig.* 760). Her blood-pressure was 160/90. She is well on a reducing diet.

Case 5.—**Calcified splenic aneurysm: incidental finding in urological investigation.**

D. H., a miner aged 31 years, presented with slight dysuria and right-sided lower abdominal pain. There were no abnormal physical signs. His blood-pressure was 140/80. Intravenous pyelography was normal except for

a calcified splenic aneurysm opposite the 12th thoracic vertebra (*Fig.* 761). It was fully calcified and measured 1·2 cm. in diameter. It lay anterior to the vertebræ in a lateral film. A mid-stream specimen of urine showed pus cells and grew *Bact. coli*. The infection responded to a course of furadantin. When last seen he had no symptoms and the urine was normal.

DISCUSSION

Owens and Coffey (1953) and others have reviewed this subject in detail, and the present discussion will therefore be brief. They found 37 patients with splenic aneurysms in 96,471 collected autopsies. The population served by the Mansfield Hospital is some 200,000. We would thus have encountered, in life, in the last five years, a ninth of the patients we serve who will sooner or later develop a splenic aneurysm. The true incidence must be far higher.

The pain in the first case described was typical of that in splenic aneurysms, and was completely relieved by operation. In these patients pain is generally considered to be due to small leaks, of which there was no evidence in this case, or to pressure on a neighbouring structure, such as the pancreas, when there may be pancreatitis (Hughes and Joske, 1955).

Many signs and symptoms have been ascribed to splenic aneurysms, and aortography has been used as a diagnostic aid (Riemenschneider, 1955, and others). Rupture presents the most dramatic picture. This has occurred in partially calcified aneurysms (Tagart, 1953), but is said never to have occurred in well-calcified ones (Von Ronnen, 1953).

The two uncalcified aneurysms in the previous paper (Ward-McQuaid, 1958) both presented with gross intraperitoneal bleeding. One survives, the lady with the porto-caval shunt whose aneurysm ruptured during pregnancy. She had a live child at a subsequent pregnancy. The other died after operation.

Allied to rupture of a splenic aneurysm is the well-documented spontaneous rupture of the splenic pedicle, particularly in pregnant women (Gallagher and Hudson, 1954).

Two years ago Mr. S. C. Anderson referred a woman to me soon after labour who had had a catastrophic intraperitoneal hæmorrhage. A rapid laparotomy did not reveal a spurting vessel, but after sucking and mopping out the peritoneum, a steady trickle of blood came from the splenic pedicle. There was no aneurysm, and the spleen itself was intact. She recovered uneventfully after removal of what proved to be a normal spleen. Another earlier patient in advanced pregnancy was operated on for gross intraperitoneal bleeding and no cause was found. She recovered uneventfully.

It would not be possible to state categorically that a spontaneous rupture of the splenic pedicle had occurred in the first patient. Recovery without finding or treating the cause is not unusual in a large series of cases of hæmoperitoneum (Ellis, Griffiths, and MacIntyre, 1958). Damage during laparotomy is not unknown, not only in an emergency operation for bleeding, but also in elective procedures such as subtotal gastrectomy. Finally, trauma before operation may not always be freely discussed, particularly if it occurred during a domestic fracas.

In the remaining 7 of the 10 patients described in this and the previous paper the splenic aneurysms were an incidental finding. In 2 they were excised, 1 with a carcinoma of the œsophagus, and 1 with a carcinoma of the kidney; both patients are well three years later. In a third patient the spleen and its aneurysm were excised deliberately because of hypertension and the size of the aneurysm, and she is also well. The remaining 4 patients (*Cases* 2–5 in the present paper) will be observed. The aneurysms have been known to be present for only 7 years, 3 years, and over a year in the last two.

There is real need for long observation of a large collected series of these patients to determine the prognosis without operation, the chances of rupture, and which aneurysms are likely to do so. It is difficult to believe that 45 per cent of them rupture, as James (1958) states in his article describing what was probably the fifteenth recovery after rupture.

Operation is often recommended and indeed practised by many surgeons treating diagnosed aneurysms reported in the literature. This has not always been so (Säfwenberg, 1937). Great numbers of calcified splenic aneurysms are being left alone without mishap even after diagnosis. Ronnen (1953) was unable to find a case of calcified splenic aneurysm in which rupture had occurred. He advised observation provided the aneurysm was asymptomatic, "the size of a cherry or smaller", completely calcified except where it joins the main trunk, and not in a woman of child-bearing age or a patient with hypertension. There is sometimes difficulty in making a confident diagnosis in calcified splenic aneurysms, and many such aneurysms are no more than incidental radiological findings in elderly people.

SUMMARY

1. A patient with severe symptoms of left-sided pain was cured by splenectomy with removal of the tail of the pancreas in which was embedded a calcified splenic aneurysm.

2. Ten splenic aneurysms have been seen in the last five years. Four patients with calcified splenic aneurysms are being observed.

3. Two patients with gross hæmoperitoneum in advanced pregnancy are discussed in relation to spontaneous rupture of the splenic pedicle.

Acknowledgements.—I am indebted to Dr. Stansfield for the histological report on *Case* 1, to my colleagues Dr. E. J. S. Townsend and Dr. L. J. Evered for the radiographs; to Dr. R. J. Twort who referred *Case* 1; and to Mr. A. McEwen Smith, under whose care *Case* 4 was admitted.

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SARCOMA OF THE URACHUS

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ONLY 1 case of this condition appears to have been reported in the last ten years. Shaw in 1949 could find only 5 cases to which to add his own, and Butler added a seventh to the literature in 1959. This report of another case may be of interest.

CASE REPORT

HISTORY.—J. H. was an only child aged 4½ years. She had been perfectly well and had developed normally until April, 1959, when her parents noticed her to be listless and pale, and the child complained of abdominal pain. Twelve days later she was admitted to hospital suffering from vague central abdominal pain and malaise.

ON EXAMINATION.—She was a pale, ill child, with a pulse of 120 and a temperature of 100·2°. Her abdomen

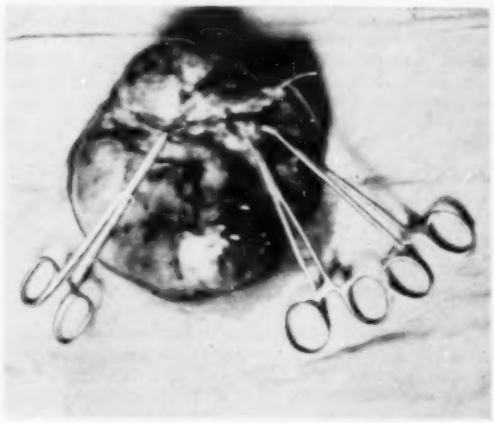

FIG. 762.—Photograph of the specimen showing artery forceps applied to the two umbilical arteries and the site of attachment of the umbilicus. A probe protrudes from the stump of the urachus.

was greatly distended by a mass which appeared to rise up from the pelvis. The abdomen was coursed by dilated veins draining radially from the umbilicus. The mass was nearly spherical, about the size of a grapefruit, and a little irregular in outline; it rose to above the umbilicus. It was firm in consistency and had a smooth surface. It was mobile from side to side but not in a vertical direction. On rectal examination the lower pole of the mass was palpable. No ascites was detected. Spleen and liver were not palpable. No other abnormality was found in the child, except a few shotty glands in the axillæ and neck.

Laboratory investigations revealed a hæmoglobin of 66 per cent and a white-cell count of 12,000 (polymorphs 74 per cent, lymphocytes 18 per cent). The blood group was A Rhesus-positive.

AT OPERATION.—A provisional diagnosis was made of teratoma of the ovary, and laparotomy was carried out. The mass was found to be almost spherical, and about 5 in. in diameter, very vascular, and situated outside the peritoneum beneath the anterior abdominal wall. It was continuous below with a pedicle containing the three umbilical ligaments in œdematous tissue, and above it was fixed to the umbilicus. The omentum was œdematous and some terminal fronds were adherent to the peritoneum covering the mass. The mass also derived some blood-supply from the dilated omental vessels. Some clear, free peritoneal fluid was present and there were numerous enlarged glands in the mesentery—swollen but soft. Other abdominal organs were normal. The tumour was removed by peeling it off the anterior abdominal wall, which it appeared not to have invaded, though the umbilicus had to be included in the excision. One inch of surrounding peritoneum was removed with the tumour. The two umbilical arteries (which were widely patent) were tied off and severed separately. A part of the bladder wall was dissected off down to the mucosa and the urachus removed intact with some bladder wall (*Fig.* 762). The defect in the bladder was repaired. The edges of peritoneum were drawn together and the abdominal wall was sewn up in layers.

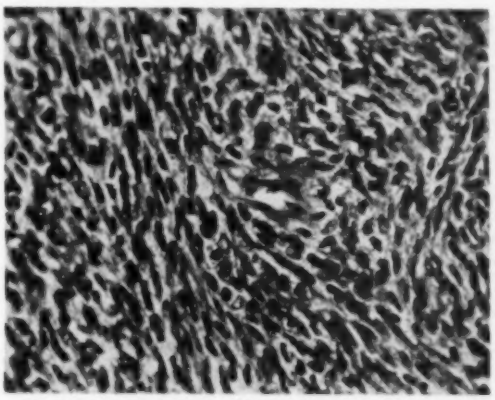

FIG. 763.—Photomicrograph of the specimen. H. & E. (× 330.)

POST-OPERATIVELY the child made a rapid recovery, having had a pint of blood before and during the operation. She left hospital on May 12, 1959, afebrile and active. On June 16 she was seen at the Royal Marsden Hospital by Professor Smithers, who found her well and with no abnormal physical signs. It was felt that it was possible that all the tumour had been removed and that post-operative irradiation would do no good and might even do harm. On June 27 she was readmitted with ascites and a central abdominal mass; by July 9 the abdominal mass had developed as large as the original lesion and the child was ill and febrile. Radiotherapy was commenced and she was given a total of 2100 r skin doses at 250 kV. by the two anterior oblique fields, over a period of 18 days. Concurrently with the radiotherapy, cyanocobalamin 1000 μg. was given daily just in case it might have been possible to induce some differentiation of the tissues of the tumour. At first there was little improvement in the size of the mass although her general condition remained poor. She required transfusion of a pint of blood for anæmia. She developed a very distended abdomen and paracentesis was performed on July 23. She became extremely emaciated and suffered continuous pain from the enormous distension of the abdomen, until she died on Aug. 6.

HISTOLOGY AND MORBID ANATOMY (*Fig.* 763).—Histology showed the tumour to be a sarcoma, and sections

through the lower end of the urachus and the portion cut off from the umbilicus and a gland removed at operation showed normal tissue only. At post-mortem the laparotomy scar was noted to have formed a keloid. The entire abdomen was filled with soft brain-like growth which was entirely retroperitoneal or intramesenteric. There was no infiltration of any abdominal organ and no metastasis was found elsewhere. The mass of secondary growth was equal to about five times the mass of the original tumour.

Dr. M. S. R. Hutt, of St. Thomas's Hospital, reported on the sections: "The tumour is composed predominantly of rather plump spindle cells arranged in whorls. There is a moderate amount of nuclear variation and occasional multinucleated cells are present. Mitotic figures are common, two or three per high-power field, and several atypical mitoses were seen. The tumour is not producing collagen, but a reticulin stain shows a fine reticulum surrounding small groups of cells.

"The tumour is infiltrating a loose normal connective tissue and in one area the tumour cells appear to be lining a canal, but no normal epithelium can be seen.

"Conclusion: This is a spindle-cell sarcoma. The overall morphological picture suggests that it may have arisen from smooth muscle cells."

Dr. N. F. C. Gowing, of the Royal Marsden Hospital, classified the tumour as an undifferentiated (embryonal) sarcoma. His report read: "The neoplasm is composed of loosely arranged spindle-shaped and spheroidal cells. The nuclei vary in shape and staining properties. Some are elongated and pachychromatic; others are spheroidal and vesicular. The cytoplasm is generally scanty and the cell boundaries are ill-defined. Mitotic activity is marked, and there are scattered giant cells containing closely-packed, hyperchromatic nuclei. A considerable number of wide vascular channels are present, but supporting stroma is otherwise scanty. Necrosis and hæmorrhage are extensive in some areas. Silver impregnation reveals abundant reticulin fibrils, which often surround individual cells to produce a fine mesh. In picro-Mallory and phosphotungstic acid hæmotoxylin preparations the cytoplasm tends to stain red and blue respectively, but a careful search fails to demonstrate any definite myofibrils or cross-striations."

DISCUSSION

Critical criteria for a diagnosis of sarcoma of the urachus were laid down by Shaw (1949) and this case fulfils them:—

1. The tumour must occupy a site consistent with the anatomical position of the urachus at the age of the patient.

2. There should be a demonstrable anatomical relationship between the tumour and the urachus.

3. The tumour must possess the pathological and histological features of a sarcoma.

Begg (1931) describes the anatomy of the urachus in great detail. He considered 6 cases of sarcoma of the urachus to have been authentic out of 7 previously reported. However, Shaw accepted only 3 of those listed by Begg and adds the cases described by Ransom (1933) and Fulle (1914) together with his own case. Butler and Rosenberg published a case report in 1959, remarking on the many similarities between their case and that of Shaw. Including this present case, 8 sarcomata of the urachus have now been described. Two of the tumours were found in adults (Grieg, 1927; Voncken and Cambresier, 1927 and seem to be different in many ways from the other six, which, as in this case, were in young children. Six cases are briefly discussed, those described by Frank, Fulle, Ransom, Butler, and Shaw, and this present case.

The youngest patient was 4 months, the oldest 11 years old. In one case the tumour was described as a rhabdomyosarcoma, one was thought likely to be a rhabdomyosarcoma, and the rest were not described as being derived from any particular cell. In 4 cases the remarkable rate of growth was commented upon—apparently exceeding the growth rate of an embryo or fœtus. This was certainly the extraordinary feature of the case described here.

Five patients died within seven months of the tumour being found, but it is encouraging to note that in the case described by Ransom, radical removal together with irradiation achieved an apparent cure, since there was no recurrence in two years. In that case the irradiation was given immediately the wound had healed, without waiting for any sign of recurrence. But this same procedure was adopted in the case of Butler and Rosenberg, without success, and they found actinomycin to be of no avail.

SUMMARY

An eighth case of sarcoma of the urachus is described in detail.

A brief discussion is made about the cases that have so far been described as occurring in children.

Acknowledgements.—I would like to express my gratitude to Mr. D. Lang Stevenson for permission to write about this case, and for his help and encouragement; to Dr. M. S. R. Hutt for commenting upon the histology and for providing the photomicrograph; to Professor Smithers for details of his treatment and observations, and for advice; and to Dr. Gowing for his histological findings and report.

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STAB WOUNDS OF THE HEART

A CASE REPORT WITH A DISCUSSION OF THE DIAGNOSIS AND TREATMENT

By B. CRYMBLE

REGISTRAR, REGIONAL PLASTIC SURGERY SERVICE, NEWCASTLE R.H.B., AND UNITED NEWCASTLE HOSPITALS

To encounter these cases has been a rarity of medical practice in this country, but there is some evidence to suggest that they might not be so uncommon in the future. Newspaper reports alone testify to an increase in the number of knife injuries in general over the past few years. More factual evidence has been given by the Home Office (1958) which reveals that known cases of felonious and malicious wounding increased by approximately 50 per cent in the period 1955–58. It is the younger criminal that uses the knife, and during this same period convictions for crimes of violence against the person have increased by no less than 125 per cent in the 17–21 years age group.

When such injuries involve the heart the mortality rate amongst those who survive the assault can be reduced appreciably by making an early diagnosis (Elkin, 1941), and planning treatment to suit the type of injury (Beck, 1942; Elkin and Campbell, 1951). This may seem to be obvious, but unexpected difficulties are often encountered in practice. The report of a recent case will illustrate the importance of these statements, and it may be timely in drawing attention to this particular problem.

CASE REPORT

A youth aged 19 years was involved in a street incident receiving a knife wound of his chest. The details are vague, but the probability is that he did not collapse until some minutes after the attack. Within half an hour he had been brought to the hospital, where he appeared to be both shocked and drunk. He was struggling and shouting incoherently, his jacket was wet with blood and vomited beer. There was a 1¼-in. stab wound of his left chest wall, anteriorly and low down, some 5–6 in. from the midline. It was neither bleeding nor sucking air at the time of examination. Probing suggested that the track was directed downwards, but palpation of the abdomen did not suggest the presence of injury below the diaphragm. Accurate examination of the heart and lungs was impossible because of his noisy behaviour but no gross changes were evident. The only diagnosis that could be made at this stage was of hæmorrhage of uncertain extent and origin, which might still be continuing internally. As a precaution an intravenous transfusion of plasma was started. His pulse was not very strong, and his movements prevented measurement of his blood-pressure. Then quite suddenly 5 minutes after his arrival he became still, his head falling back, with his mouth open and his eyes staring. Breathing appeared to have ceased and there was

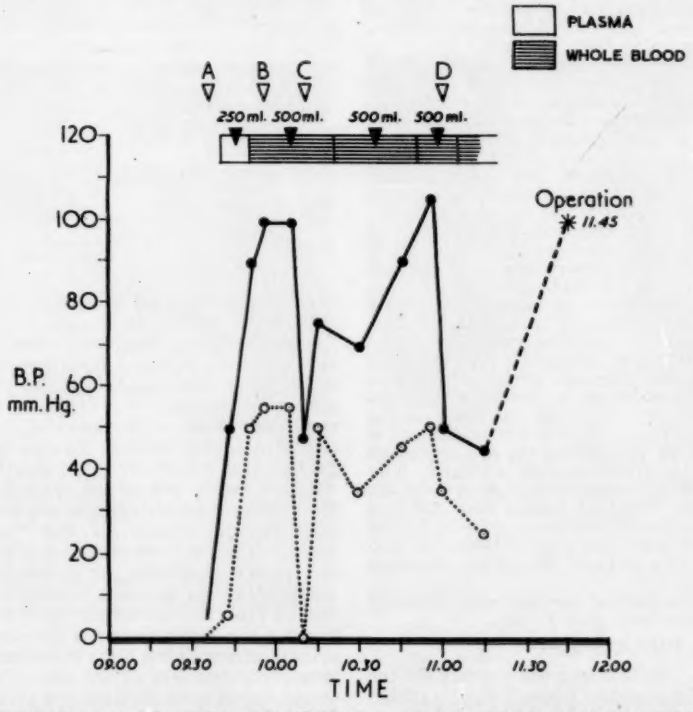

FIG. 764.—Chart showing the course of the systolic and diastolic blood-pressures between the time of admission and the time of operation.

no evidence of a heart-beat. It was as though he had died (*Fig.* 764, A). Within one minute, however, shallow but spontaneous respiration appeared and a very weak pulse became palpable. The transfusion was now changed to blood and after 10 minutes of steady improvement he had a blood-pressure of 100/50 mm. Hg (*Fig.* 764, B) and he was talking. This remarkable reversal in his condition was so unexpected that the incident was thought to be the effect of only minor trauma in an intoxicated subject. The noisiness had returned with consciousness and again made a satisfactory chest examination impossible. After 15 minutes of continued improvement he suddenly collapsed once more, showing an extreme fall in blood-pressure and again losing consciousness (*Fig.* 764, C). The rate of the transfusion was increased and recovery followed within a few minutes. Further progress was slow but steady, and the effects of alcohol gradually became less obvious. Arrangements were being made to transfer him to the theatre from the Casualty Department when, at 11.0 p.m., he collapsed for the third time (*Fig.* 764, D). Now the signs of bleeding were very evident despite the previous transfusion of 1500 ml. of blood, and he began to complain of severe epigastric pain. Palpation revealed extensive upper abdominal tenderness, with guarding almost to the degree of rigidity. It was decided that a delayed intra-abdominal hæmorrhage was in progress, possibly occurring from the liver or spleen, and it was agreed to perform a laparotomy rather than to explore the wound.

In the theatre through an upper abdominal midline incision the peritoneal cavity was opened and found to be devoid of free blood and the diaphragm was intact.

A left anterior thoracotomy which included the stab wound was then performed and in the pleural cavity was found a large quantity of partly clotted blood. Also a tense hæmopericardium was to be seen, with a blood-clot pouting from an anterior laceration. Opening of the pericardial sac and evacuation of the blood allowed the compressed heart to beat vigorously. A further clot of blood which was seen occluding a penetrating wound of the anterior wall of the left ventricle was suddenly blown out and followed by a forceful gush of blood. This hæmorrhage was readily controlled by finger pressure and the hole was sutured. When the residual blood had been cleared from the pericardial sac and pleural cavity, the chest was closed. A total of 17 pints of blood had been transfused from the time of his arrival 3½ hours previously.

The post-operative recovery was rapid and uneventful and he was discharged home after 12 days.

DISCUSSION

1. Diagnosis.—In stressing the urgency for the early recognition of a cardiac injury Elkin (1941) has felt it necessary to list several clinical features which have more than once misled even the experienced, thereby causing considerable delay in diagnosis, which in some cases had unfortunate consequences.

These features included:—

a. An intoxicated patient talking incessantly and unintelligibly.

b. An interval of time between the injury and a subsequent collapse.

c. A shocked patient responding dramatically to a small transfusion of blood.

d. A wound that does not suck air.

e. A wound that does not appear to be directed towards the heart.

f. The absence of any signs of venous congestion.

It will be noted that every one of these was observed in the case reported above. In retrospect, not one person who saw this case prior to operation noticed any signs of venous congestion although gross tamponade was demonstrated later, and presumably its absence was the result of the severe depletion of circulating blood-volume by the hæmorrhage. The soundness of Elkin's advice to regard all wounds in possible situations as involving the heart until proved otherwise is clearly illustrated by this case.

2. Treatment.—The line of treatment to follow once the injury has been diagnosed constitutes a subject of much controversy in America, where most of these cases are encountered. At one time all survivors of the injury were treated by immediate operation. The operative mortality rate, however, was very high, being 22 per cent in Elkin and Campbell's (1951) series treated between 1941 and 1944. Such results stimulated a revival of interest in the more conservative method of pericardial aspiration, which had long ago been advocated by Napoleon's surgeon, Baron Larrey. After 1944 these same authors therefore treated a series of 18 cases by this method, with an overall mortality rate of approximately 6 per cent. They tabulated their cases thus:—

Grade I	Minimal hæmopericardium	3 cases	0 deaths
Grade II	Large non-progressive hæmopericardium	9 cases	0 deaths
Grade III	Progressive hæmopericardium	5 cases	1 death
Grade IV	Hæmopericardium and hæmothorax	1 case	1 death

Many other surgeons were able to report a similar striking reduction in the mortality rate by employing this alternative treatment, but a few did not share this experience. A study of Elkin and Campbell's cases suggests an explanation for these opposing views. The majority of the cases had the less severe injuries and responded well to treatment by aspiration, the 2 deaths occurring amongst the minority in the severer grades. It is interesting to see that the surgeons who did not favour pericardial aspiration practised in those areas where the injuries are usually inflicted by such vicious weapons as the 'crab-apple switch knife'. The wave of enthusiasm for treatment by aspiration is justifiable, but it must not be allowed to detract from the fact that there is a definite though limited place for immediate operation. The indication for the latter course is the domination of the clinical picture by the signs of hæmorrhage. To have attempted treatment of the case cited by aspiration would almost certainly have had a fatal outcome.

SUMMARY

A case of a stab wound of the heart is described, with particular reference to the unexpected difficulties that may be encountered in diagnosis. A suggestion is made as to the indications for immediate operation as the definitive treatment, rather than pericardial aspiration.

I should like to thank Mr. A. Hedley Whyte for permission to publish this case.

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IDIOPATHIC CHYLOTHORAX

TWO CASE REPORTS

By KINGSLEY LAWRANCE*

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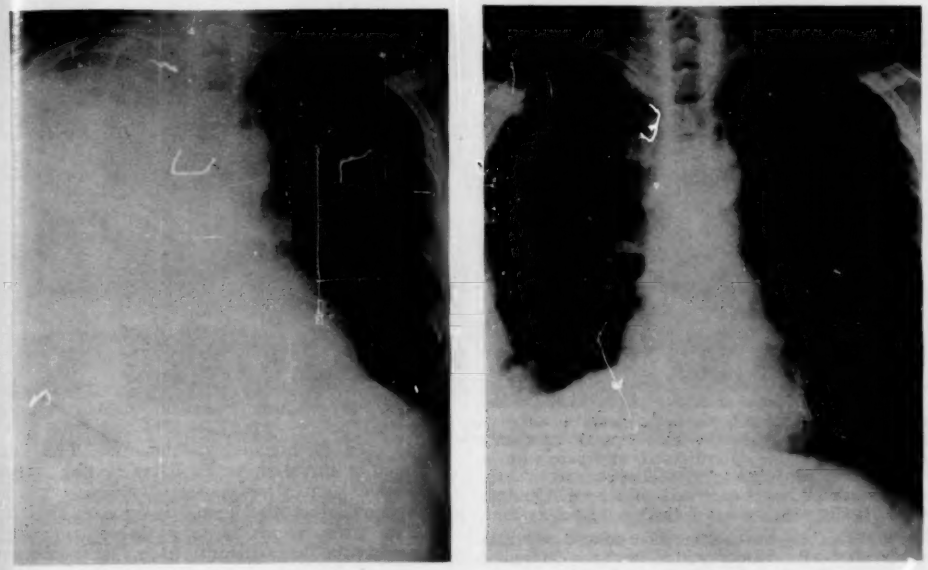
THE aetiology of chylothorax has been subjected to various forms of classification (Hoffman, 1957; Nix, Albers, Dugas, and Wendt, 1957) and the correct name for the accumulation of chyle in the chest without any apparent cause is open to some doubt. Often called 'spontaneous', it should perhaps more correctly be called 'idiopathic chylothorax'. The treatment of chylothorax (regardless of aetiology) has been equally variable (Nix and others, 1957), but the current consensus seems to be in favour of ligation of the thoracic duct. From a recent survey of the literature, Garamella (1958) quotes 40 cases of traumatic chylothorax treated by thoracic duct ligation, and brings the total to 41 by adding a case of his own; no mortality has been reported.

In neither of the two cases reported here was any cause established for the chylothorax; there was no history of injury and no condition was present which might have led to the collection of chyle in the chest.

CASE REPORTS

Case 1.—A 26-year-old male in 1952 (at the age of 21 years) developed fatigue, periorbital œdema, and swelling of the ankles. In November, 1952, he had acute appendicitis and the appendix was removed. Early in 1953 the symptoms of fatigue and œdema recurred and he was found to have hypo-albuminæmia; other studies at this time, all of which were normal, included chest fluoroscopy, I.V.P., G.I. series, and barium enema, E.C.G., blood-film, liver function tests, and urinalysis. Later in the same year a G.I. series showed some irregularity of the terminal ileum and subsequent exploration revealed adhesion and an area of enteritis 6 in. long in the jejunum.



A B

FIG. 765.—*Case* 1 before and after operation.

The œdema and fatigue persisted, but in May, 1957, he developed chest pain and fever; a chest radiograph showed fluid in the right chest (*Fig.* 765 A) and aspiration revealed chyle. A bronchoscopy at that time was normal. In August, 1957, he had thrombophlebitis in the right arm.

In January, 1958, the hypo-albuminæmia was investigated further and the iodinated albumin turnover study revealed a very rapid degradation of iodinated albumin with a five times normal level of iodine in the stool; there was also an increased level of iodinated polyvinyl pyrrolidine excretion in the stool. Since the presence of chyle had first been demonstrated, repeated and large thoracenteses had been carried out but the chyle always re-accumulated; his general condition remained good and the physical signs were confined to those of fluid in the right chest. In April, 1958, almost a year after the chylothorax had first appeared, a thoracotomy was carried out (K. L.); at the time of operation there were about 3000 c.c. of chyle in the right chest and the visceral pleura was thickened over all lobes. The chyle seemed to be coming from just above the diaphragm, in the space between the aorta, azygos vein, and diaphragm;

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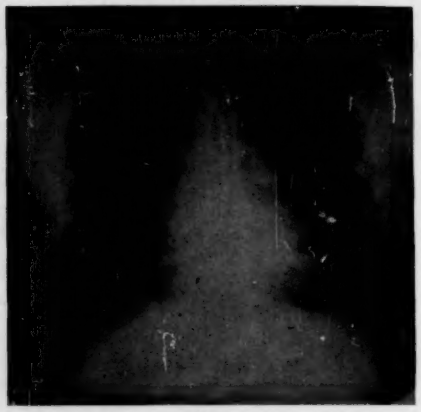
the thoracic duct was dissected out in this region and cannulated; an injection of 1 c.c. of indigocarmine distally showed no leak below the point of cannulation; the duct was therefore ligated and a portion removed; histology later confirmed that this was normal thoracic duct. The greater part of the thickened visceral pleura was removed. His post-operative course was uneventful, but because of the length of time the condition had been present, the lung did not re-expand quite fully (*Fig.* 765 B); however, there was no recurrence of chyle in the pleural space.

A



B

FIG. 766.—*Case* 2 before and after operation. Note the better result obtained with earlier surgery in comparison with the post-operative films of *Case* 1 (*Fig.* 765 B).

It had been thought that the hypo-albuminæmia might be related to the enteritis seen at laparotomy in 1953; therefore, in July, 1958, the abdomen was re-explored; the small bowel appeared quite normal, but an unexpected finding was 500 c.c. of chyle in the peritoneum.

Case 2.—This 22-year-old male first noticed shortness of breath six weeks before admission; this increased gradually and he also developed an irritable cough. Immediately prior to admission the dyspnœa became severe and incapacitating. There was no past history of ill health or weight loss; and there was no recent history of any injury whatsoever. On physical examination his general condition was good and the only abnormal signs were those of fluid in the right chest. Radiographs confirmed the presence of fluid (*Fig.* 766 A) and showed no abnormal lung changes or evidence of hygroma. The chest was aspirated on several occasions over this six-week period and about 2500 c.c. of chyle were removed; the chyle, however, always re-accumulated.

At operation (J. A. A.) the right chest was opened and found to contain about 2750 c.c. of chyle; this was in spite of 1000 c.c. having been removed at thoracentesis the day previously. The pleura was red and œdematous, but there were no deposits of fibrin and the lung appeared healthy. No source of the flow of chyle could be located, even after the introduction of cream into the stomach; a small pleural diverticulum under the vena azygos was examined especially, but with negative results. The thoracic duct was therefore explored immediately above the diaphragm; after it had been dissected out, a portion of it was removed between ligatures. Histological examination later confirmed that this was normal thoracic duct. His post-operative course was uneventful and the lung expanded completely (*Fig.* 766 B); there was no recurrence of chyle either during the post-operative period or six months later.

COMMENT

The second case was more straightforward and shows the advantage of prompt treatment in so far as the normal function of the chest is more readily restored (*Figs.* 765 B, 766 B). In the first case no relationship could be established between the hypo-albuminæmia and the chylothorax, and no such association could be found from a survey of the literature: however, the possible relationship of the two conditions will be discussed by Waldman and Gordon (1961) in a forthcoming publication. The finding of chyle in the peritoneum is interesting; whether the chylous ascites was present all along or only since the thoracic duct was ligated is open to conjecture, but the latter seems more probable; since this is apparently the first patient to have a laparotomy after ligation of the thoracic duct, it remains to be seen whether or not chylous ascites is a complication of this procedure.

SUMMARY

Two cases of idiopathic chylothorax treated by thoracic duct ligation are reported; the advantage of early treatment and the possibility of chylous ascites being a complication of thoracic duct ligation are commented upon.

I am indebted to Dr. John M. Potter for the report on the findings at laparotomy, and to Mr. John A. Aylwin for permission to report *Case* 2. *Case* 1 was seen and operated on while I was Visiting Scientist to the National Heart Institute, Bethesda, Maryland, U.S.A.

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A SURGICAL APPROACH TO MITRAL INSUFFICIENCY

By C. N. BARNARD, M. B. McKENZIE, and V. SCHRIRE, Cape Town*

Despite the remarkable advances in cardiac surgery, a suitable operation for the correction of all pathological types of mitral regurgitation has not yet been established. Many ingenious blind techniques have been devised and enthusiastically advocated for the surgical correction of mitral regurgitation in the recent past (Bailey, Jamison, Bakst, Bolton, Nichols, and Gerinhardt, 1954; Blalock and Johns, 1954; Davila, Glover, Trout, Mansure, Waad, Janton, and Ioia, 1955; and Kuykendall, Ellis, and Grindlay, 1958), but success was invariably transitory and such operations were short-lived.

The trend is now towards surgical treatment of mitral regurgitation by open heart surgery. Surely an intelligent attempt at correction can be made only when the valve is adequately visualized and the various pathological factors appraised, evaluated, and corrected under direct vision, employing a mechanical pump oxygenator?

Before attempting the correction of mitral regurgitation, the surgeon should acquire a clear, three-dimensional concept of the anatomy of the mitral valve region, a sound knowledge of the normal mechanism of mitral valve closure, and also the lesions which may cause the derangement of this mechanism.

Anatomy of the Mitral Valve.—Anatomically, the mitral valve mechanism consists of four main parts: the annulus fibrosus, the valve leaflets, the chordæ tendineæ, and the papillary muscles. It is not within the scope of this paper to discuss the anatomy in detail, but certain observations which concern surgery in this region must be mentioned.

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The mitral valve is a flat cone (Van Der Spuy, 1958). This cone has as its base the mitral annulus, on which hinge the two cusps which form the sides of the cone. As the anteromedial half of the mitral annulus is closely related to the posterolateral half of the aortic root and the corresponding two aortic cusps, it is thus biconcave. The anteromedial mitral cusp, being hinged in this position, forms an integral part of the left ventricular outflow tract during systole.

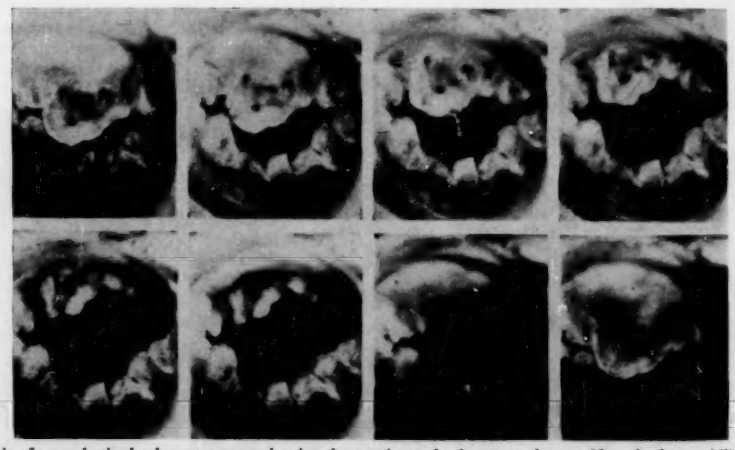


Fig. 767.—Cycle of normal mitral valve movement, showing the opening and subsequent closure. Note the free mobility of the anteromedial leaflet, and that the posterolateral leaflet only provides an impassive ridge against which the anteromedial cusp can close.

The posterolateral portion of the ring is formed by the common fibrous origin of the posterolateral cusp, the corresponding part of the left atrium, and the base of the left ventricle (Van Der Spuy, 1958). This portion of the mitral ring is related to the terminal branches of the circumflex coronary artery, the proximal portion of the great cardiac vein, and the coronary sinus.

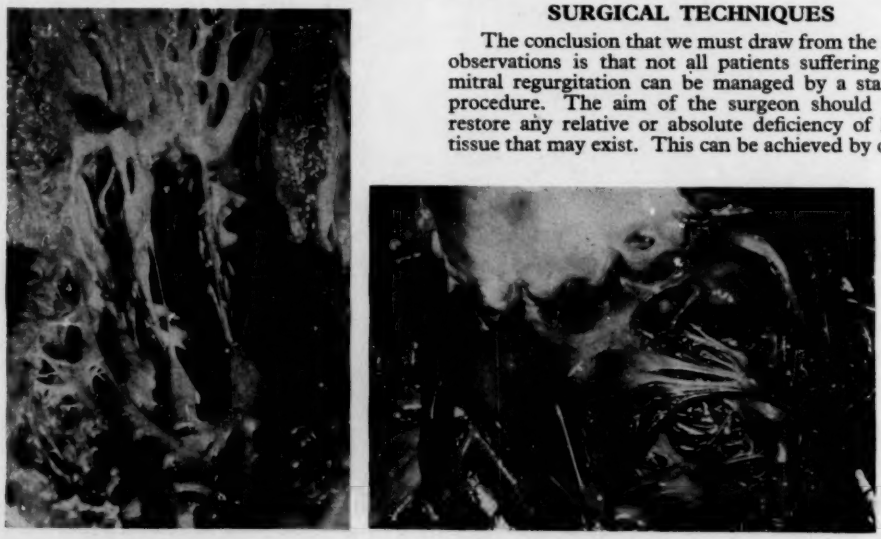
Normal Mitral Valve Closure.—Chiechi, Lees, and Thompson (1956), and others (Brock, 1952; Bailey and Bolton, 1956; Harken and Block, 1955; Little, 1951; and Van Der Spuy, 1958) have attempted to describe in detail the movements and functions of the various components of the mitral valve mechanism. For practical purposes, the valve orifice is guarded by two leaflets which constitute a flap-type valve, activated by the differences in hydrostatic pressure between the atrium and the ventricle, and passively restrained by their anchoring chordæ and papillary muscles.

The anteromedial cusp is much longer than the posterolateral one, and the chordæ are attached only to the peripheral zone of the cusp, leaving a triangular area at the base, which is much thinner, free to billow towards the cavity of the left atrium during systole. The chordæ of the posterolateral cusp are inserted more widely on its ventricular surface, leaving

only the basal edge free to bulge forwards during systole.

It can thus be seen that normally the closure of the mitral valve orifice is mainly effected by the freely mobile anteromedial leaflet, and that the posterolateral leaflet only provides a ridge or shelf against which the anteromedial cusp can abut to effect closure (*Fig.* 767).

The annulus has a definite sphincter-like action (Brock, 1952; Van Der Spuy, 1958). This action decreases the size of the valve orifice and tends to approximate the two major leaflets during systole by diminishing their range of excursion before closure is effected.



A B

FIG. 768.—A, Photograph of a congenital cleft of the anteromedial leaflet of the mitral valve, as seen in endocardial cushion defects. Note the abnormal chordal attachments at the side of the cleft. B, Photograph of an acquired perforation of the anteromedial leaflet of the mitral valve, following acute bacterial endocarditis. Both these defects caused mitral regurgitation.

Pathology of Mitral Regurgitation.—The clinical picture of mitral regurgitation may result from numerous widely differing pathological lesions, the essential cause being a deficiency of leaflet tissue, either absolute or relative, or a combination of both.

An absolute deficiency of leaflet tissue may result from a cleft or perforation of the cusps, either congenital or acquired (*Fig.* 768), but more commonly from rheumatic scarring of the cusps. The posterolateral leaflet of the mitral valve is more susceptible to the ravages of the rheumatic process. However, this may be more apparent than real, because of the greater length of the anteromedial cusp. An equal extent of rheumatic involvement of both leaflets from the free margin basally will involve only one-third of the anteromedial leaflet, while the posterolateral cusp will be rendered almost completely rigid. In such cases, although the middle and basal thirds of the anteromedial leaflet remain thin and flexible, the shelf provided by the posterolateral leaflet is lost. Mitral regurgitation thus results.

A relative deficiency may be due to dilatation of the annulus or to changes that occur in the junctional tissues, such as thickening and shortening, or fusion of the chordæ, with a resultant limitation of cusp mobility.

In addition, in most cases of mitral insufficiency, some degree of functional regurgitation is probably superadded as a result of compensatory left ventricular hypertrophy, and later dilatation, which may occur.

SURGICAL TECHNIQUES

The conclusion that we must draw from the above observations is that not all patients suffering from mitral regurgitation can be managed by a standard procedure. The aim of the surgeon should be to restore any relative or absolute deficiency of leaflet tissue that may exist. This can be achieved by one or more of the following techniques, depending on what pathological changes are encountered at open operation: (1) Repair of cleft or hole which may be present in mitral leaflets; (2) Mobilization of the anteromedial cusp; (3) Provision of a shelf against which the anteromedial cusp can close.

1. Repair of Cleft or Hole in Mitral Leaflets.—A hole in the mitral valve, either congenital or acquired, can be corrected by simple suture of the defect. However, the correction of mitral regurgitation from a cleft anteromedial leaflet, as found in endocardial cushion defects, needs more than simple suture as described by Cooley, Kirklin, and Horschborger (1957), Cooley, Latson, and Keats (1958), McGoon, Dustane, and Kirklin (1959), and Barnard, Phillips, De Villiers, Casserley, Hewitson, Van Der Riet, and McKenzie (1959).

As pointed out in the section on the anatomy of the mitral valve, the chordæ of the anteromedial leaflet are attached only to the periphery of the cusp, allowing free mobility of the central trigone. In endocardial cushion defects there are abnormal chordal attachments which often extend along the cleft up to the annulus (Edwards, 1960) (*see Fig.* 768 A). These abnormal chordal attachments prevent free mobility of the trigone, and therefore, even after careful

suturing of the cleft, regurgitation persists as the leaflet is still immobile.

To obtain complete correction of the regurgitation in these defects, it is necessary to divide the abnormal chordal attachments and completely mobilize the trigone of the anteromedial leaflet before the cleft is sutured. This can be done without any danger of eversion of the cusp during systole, as the normal chordæ at the periphery will prevent this.

2. Mobilization of Anteromedial Cusp.—As the anteromedial cusp is the 'main flap' in the closure of the mitral orifice during systole, free mobilization of this leaflet is of the utmost importance in the correction of rheumatic mitral regurgitation. To achieve this, three avenues can be explored: (*a*) Separation of fused commissures; (*b*) Removal of calcium from the leaflet; (*c*) Treatment of subvalve changes interfering with cusp mobility.

a. Separation of Fused Commissures.—Although commissural separation, either by blunt or by sharp dissection, may increase the mobility of the anteromedial cusp, this manœuvre may increase regurgitation if performed when there is annular dilatation. In these circumstances the fusion of two leaflets at the commissures acts as an annuloplasty (*see below*), and when this restraint is broken the posterolateral portion of the mitral annulus will move farther away and thus increase the distance which the anteromedial cusp has to extend to close the valve.

It appears, therefore, that commissurotomy can be of value only when the annulus is of normal size, or smaller than normal.

b. Removal of Calcium from the Anteromedial Leaflet.—Free mobility of this leaflet may be prevented by plaques of calcium which prevent the 'trigone' of the valve from billowing towards the cavity of the left atrium. Once these masses of calcium are broken up by gentle crushing and they have been removed by picking the pieces out of the leaflet, it will swing freely, like a great sail, in response to the hydrodynamic stresses usual in this region.

c. Treatment of Subvalvular Changes preventing Cusp Mobility.—An attempt should be made to separate abnormal chordopapillary fusions. Direct pressure with a finger-tip may separate some adhesions, but it may be necessary to separate the papillary muscles by sharp dissection. In some cases the shortened, retracted chordæ of the posterolateral leaflet can be divided, allowing more mobility of the chordæ of the anteromedial leaflet. This will not cause eversion of the posterolateral cusp during ventricular systole, as severe fibrosis has made this cusp a passive ridge.

3. Provision of a Posterolateral Shelf against which the Anteromedial Cusp can close.—The importance of the provision of a posterolateral shelf in the treatment of mitral regurgitation has been recognized by many workers. Substances such as pericardial tubes (Moore and Shumacker, 1953), vein grafts (Henderson and Law, 1953), and tendon grafts (Glover, Henderson, Martutti, and Gregory, 1952) have been placed in the posterior commissure to provide such a shelf in the area where regurgitation is maximal. Ridged Ivalon sponge (DeWall, Warden, Lillehei, and Varco, 1956; and

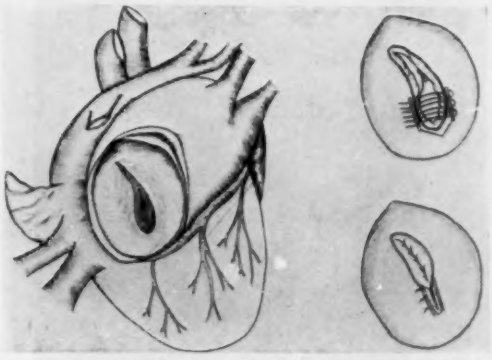
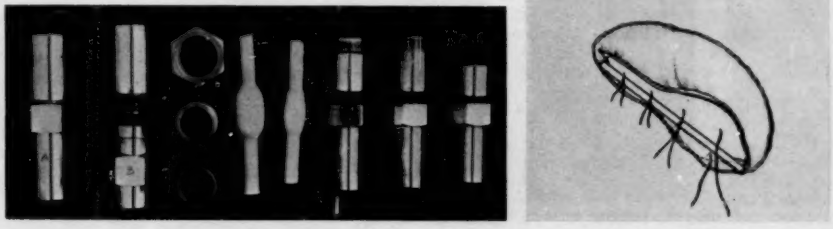


FIG. 769.—Diagram illustrating the annuloplasty operation.



A B

FIG. 770.—A, Photograph of the metal moulds and the moulded dumb-bell-shaped immobile prosthesis. B, Diagram illustrating the insertion of a dumb-bell-shaped immobile Ivalon prosthesis to provide a posterolateral buttress.

Glenn, Gentsch, Hume, and Guilfoil, 1956), a mobile Ivalon sponge sling (Johns and Blalock, 1954), and a baffle of lucite (Harken, Block, Ellis, and Dexter, 1954) have been placed in the mitral orifice with the same purpose in mind. Approximation and elevation of the posterior aspect of the mitral valve will have the same effect, and this has been attempted with the use of pericardial strips (Bailey and Bolton, 1954), vein slings (Sakakibara, 1955), and silk sutures (Kay and Cross, 1955; and Nichols, 1957). The use of an artificial cusp in place of the posterolateral leaflet was suggested by Brock in 1952, and reported by Gott, DeWall, Gonzalez, Hodges, Varco, and Lillehei in 1957. All these procedures have the same common aim in that they attempt to provide a buttress against which the anteromedial cusp can meet and close during ventricular systole.

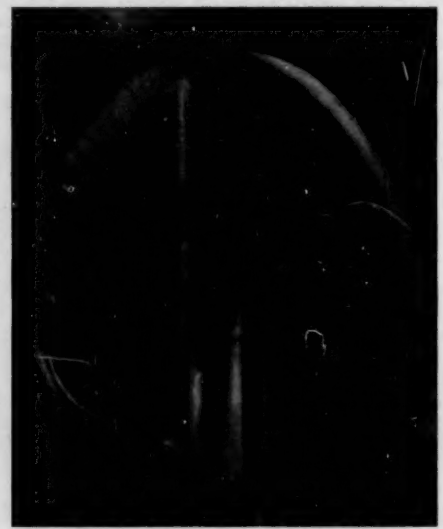


FIG. 771.—The mobile Silastic prosthesis.

If regurgitation persists after the surgeon has fully mobilized the anteromedial leaflet, it means that the anteromedial flap is still not capable of moving across the valve orifice to close against the posterolateral shelf, and leaking can be stopped or lessened now only by providing such a shelf. An improvement in valve function in these conditions can be achieved either by bringing the existing shelf nearer to the anteromedial cusp, or by building an artificial posterolateral ridge.

a. Annuloplasty is the best operation for approximating the anteromedial cusp and the posterolateral buttress. This is the operation of choice when annular dilatation is the main cause of the separation, and when there is not much loss of posterolateral leaflet substance.

This operation for the correction of mitral regurgitation was reported by Gott and others (1957), Lillehei, Gott, DeWall, and Varco (1957) and Kay, Nogueira, and Zimmerman (1958.) In performing an annuloplasty care must be taken when placing stitches to avoid the important structures right around the mitral annulus (*see* 'Anatomy' *above*). The stitches must be placed in the annulus and securely tied over pillows of compressed Ivalon sponge, to prevent them tearing out at a later date (*Fig.* 769).

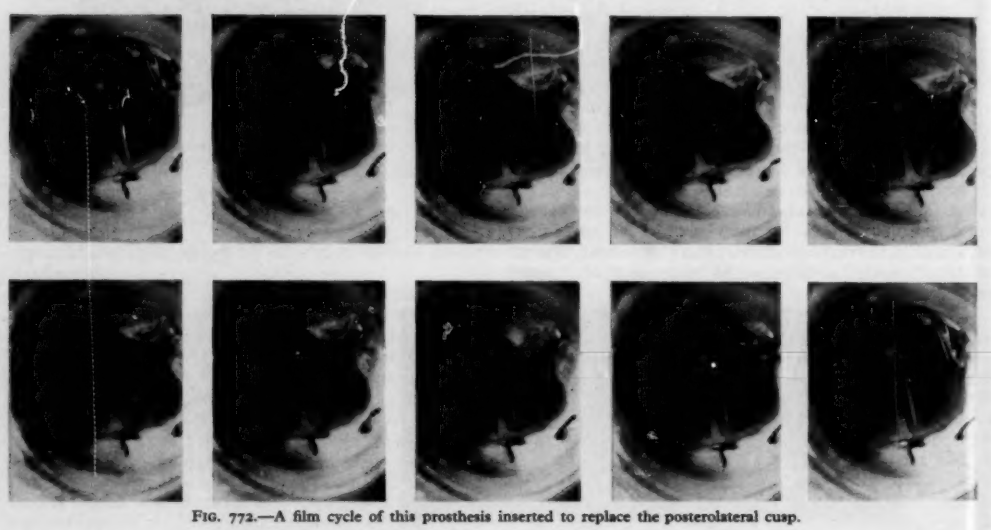


FIG. 772.—A film cycle of this prosthesis inserted to replace the posterolateral cusp.

b. Building a Posterolateral Ridge is the procedure of choice when the valve ring is normal in size or smaller than normal, and when the inability of the anteromedial leaflet to close the valve is due to extensive destruction and immobilization of the posterolateral cusp. The function of this useless cusp can be replaced by an immobile or a mobile prosthesis.

i. *Immobile prosthesis*: Gott and others (1957) and Lillehei, Gott, DeWall, and Varco (1958) reported the

use of an immobile prosthesis of compressed polyvinyl sponge. Cylindrical metal moulds of several diameters were fabricated and an assortment of polyvinyl cylinders prepared and sterilized. At operation, a cylindrical prosthesis of suitable size was selected and, by means of three or four vertical mattress stitches of No. 2 silk, secured against the ventricular surface of the posterolateral portion of the mitral annulus. In this way a buttress is provided against which the aortic mitral leaflet can close.

We prefer the use of dumb-bell-shaped compressed Ivalon prostheses for the same purpose (*Fig.* 770). These have the advantage that the prosthesis can be so placed that the wider portion of the dumb-bell is situated at the area where the separation of the two cusps is greatest, and the thinner portion where the separation is not so great, thus minimizing the possibility of stenosis in this area.

ii. *Mobile prosthesis*: Diseased valves are encountered where, after an adequate shelf is provided to correct the regurgitation by means of an immobile prosthesis, stenosis has resulted. In these cases a mobile prosthesis is needed which will not interfere with the valve orifice during diastole, but which will bulge up during systole to provide a posterolateral shelf. Such a prosthesis has been developed in our laboratory and will be the subject of another paper (*Figs.* 771, 772).

CASE REPORTS

During the past year, at Groote Schuur Hospital, Cape Town, several patients with advanced forms of mitral valve disease with regurgitation have been operated upon. Three of these cases are reported below to illustrate the various surgical techniques suggested.

Case 1.—**Dominant mitral stenosis with mild regurgitation.—**

I. K., a coloured woman aged 29 years, first attended in 1953 with effort intolerance progressing almost to total disability and angina pectoris of 18 months' duration. There was a childhood history of rheumatic fever. Peripheral cyanosis and polycythæmia with a packed-cell volume of 62 was present. The pulses were small (B.P. 125/95) and the jugular venous pressure moderately elevated, with a dominant A wave. The apex was not displaced, but the pulsation was right ventricular with a lift over the outflow tract, a pulmonary ejection sound, an early diastolic murmur, and right ventricular triple rhythm, all indicative of severe pulmonary hypertension. A grade 3/6 pansystolic murmur at the apex was followed by a moderately loud rumbling mid-diastolic and presystolic murmur. The electrocardiogram showed marked right ventricular hypertrophy and the radiograph showed severe right ventricular enlargement, moderate left atrial enlargement, and gross pulmonary arterial dilatation with peripheral pruning. The mitral valve was calcified.

At operation the mitral valve admitted the distal phalanx (2 sq. cm.—Schrire, Vogelpoel, Phillips, and Nellen, 1955), and was slightly incompetent. After dilatation, a valvotome was inserted, splitting the posteromedial commissure only. Although the final result was inadequate, with a 4 sq. cm. orifice, the left atrial pressure was halved.

Within two months of operation the patient's effort tolerance had considerably improved and the anginal pain had disappeared. The physical signs, however, were completely unchanged.

Improvement continued and was maintained for 18 months without any alteration in the physical signs, electrocardiogram, or radiograph. She then developed bronchitis and for the first time a tricuspid systolic murmur appeared. A year later, following a recurrence of bronchitis, effort dyspnœa reappeared. In May, 1956, a recurrence of bronchitis precipitated cardiac failure. Thereafter recurrent attacks of bronchitis and pneumonia were associated with progressive deterioration of cardiac function and, with the onset of atrial fibrillation, her condition was as bad as before operation. Tricuspid valve murmurs were present, in addition to the signs of

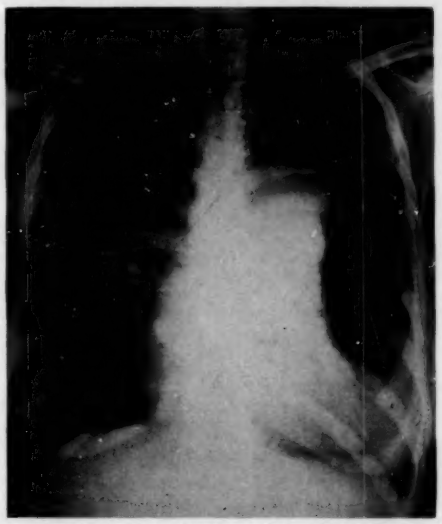


FIG. 773.—Radiograph in *Case* 1 which shows aneurysmal enlargement of the main pulmonary arteries with peripheral pruning. Slight cardiomegaly is present and the enlarged left atrium forms part of the right border of the heart. Left pleural thickening is present.

mixed mitral valve disease and of severe pulmonary hypertension. The electrocardiogram still showed right ventricular hypertrophy with atrial fibrillation and the pulmonary arteries were now aneurysmal (*Fig.* 773).

In August, 1959, the mitral valve was explored with a cardiac by-pass. The left atrium was hypertrophied and dilated, and the mitral valve orifice narrowed to 1 cm. diameter. There was no dilatation of the mitral annulus, but there was severe shortening of both mitral leaflets, with calcification of the cusps, especially the anteromedial one. The two fused commissures were divided with a knife up to the annulus, which gave adequate correction of the stenosis, but the valve was now grossly regurgitant and there was no mobility of the leaflets because of the severe calcification. The anteromedial leaflet was mobilized by the crushing and removal of the calcium, providing a very good functioning anteromedial leaflet, which compensated for the shortened posterolateral leaflet and corrected the regurgitation. The change in pressure contours is shown in *Fig.* 775. The subsequent course to date, 18 months after surgery, has been satisfactory, with complete disappearance of the symptoms and the signs for the first time have altered. A grade 3 pansystolic murmur at the apex is now associated with a short mid-diastolic murmur only. The right ventricular gallop has disappeared and the signs of severe pulmonary hypertension are somewhat reduced. The atrial fibrillation, however, could not be corrected.

Case 2.—**Dominant mitral regurgitation with mild stenosis.—**

J. F., a white woman aged 44 years, was first seen on Oct. 1, 1958, with a story of recurrent attacks of rheumatic

fever as a child, and established heart disease by the time she was 30. Minimal symptoms developed, but by 1947 she was thought to be sufficiently disabled to have a mitral valvotomy performed elsewhere. This was followed by progressive deterioration in effort tolerance. Hysterectomy was performed in 1955, following a pregnancy. Pulmonary embolism then occurred and the inferior vena cava was tied. Her symptoms progressed until finally she was admitted in congestive cardiac failure, with atrial failure and mitral stenosis and regurgitation. Retrograde angiocardiography excluded significant aortic regurgitation. Nine months later she developed a right hemiplegia and at the time of admission she was dyspnœic, as well as dysphagic and hemiplegic. She was now able to walk only a few yards because of dyspnœa, and was having frequent attacks of pulmonary œdema. Intensive and prolonged treatment with diuretics and digitalis had failed to alter the progression of her symptoms.

FIG. 774.—Radiograph in *Case* 2, which shows the gross cardiomegaly due mainly to aneurysmal enlargement of the left atrium and left ventricular enlargement. Pulmonary congestion is present, and the aneurysmal dilatation of the pulmonary veins produces a prominence on the mid-left border of the cardiac shadow.

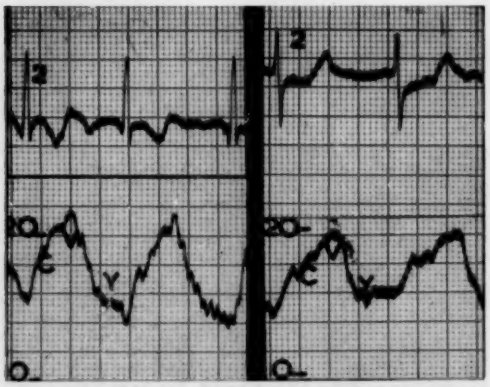


FIG. 775.—Tracing (*Case* 1) to show a moderately elevated left atrial pressure (L.A.P.) with a CV wave, suggesting some valvar regurgitation. The Y descent is slow and continuous, indicating stenosis. After operation the main change has been in the Y descent, which is now normal.

The pulse was small, the blood-pressure normal, the jugular venous pressure moderately elevated, and hepatomegaly was present. The apex beat was in the sixth space in the anterior axillary line, with a diastolic thrill. There was a pansystolic grade 3/6 murmur at the apex, a third heart-sound, and a grade 2/4 mid-diastolic murmur. A delayed opening snap was present at the base. The electrocardiogram showed digitalis effect, probably on left ventricular hypertrophy. An aneurysm of the left atrium (*Fig.* 774) with systolic expansion was present, with left ventricular enlargement and well-marked calcification of the mitral valve.

At operation in November, 1959, using a cardiac by-pass, a giant left atrium with systolic expansion was present; a systolic thrill was palpable at the back of this chamber and there was aneurysmal enlargement of the pulmonary veins. Mitral regurgitation was found, but no real stenosis, the mitral valve being about 4 cm. in diameter; the anteromedial cusp was freely mobile and without calcification; the commissures were only slightly fused, with slight calcification at the site of the medial commissure. Regurgitation was due to the loss of leaflet substance of the posterolateral cusp, with shortening of the chordæ. There was also slight aortic regurgitation. As annuloplasty was not possible, an Ivalon baffle was positioned in front of the posterolateral cusp with three interrupted No. 1 silk mattress sutures. These sutures were tied over a strip of Ivalon, which caused some narrowing of the mitral valve, but it remained 2½ cm. in diameter. There was still some regurgitation at the medial commissure, but there was no way of correcting this. The alteration in pulse contour is shown in *Fig.* 776.

The post-operative course was extremely stormy, the main difficulty being respiratory and not cardiovascular. The rigid œdematous lungs prevented proper respiration, so that tracheostomy was necessary with intermittent positive pressure. The response was most rewarding, although progress was slow. By the time of discharge a month after operation, she had improved considerably from the symptom point of view; there was less evidence of left ventricular overactivity though the murmurs were not greatly changed. A year later she had remarried and was living a normal life.

Case 3.—**Pure mitral regurgitation.**—

V. L., a white woman aged 30 years, developed valvular disease of the heart at the age of 6 years, following an attack of rheumatic fever. At school she did not take part in games, and when she was 17 years old she presented with Osler's nodes and a soft apical systolic murmur, though there were no symptoms referable to the heart. Penicillin therapy was successful in controlling the infection. A year later, subacute bacterial endocarditis recurred and thereafter she had two more attacks, each proven by a positive blood-culture and each responding

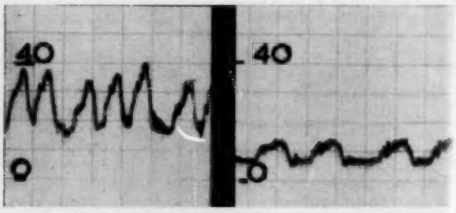


FIG. 776.—Tracing (*Case* 2) to show a markedly elevated L.A.P. with a CV wave of mitral regurgitation and rapid Y descent. After operation there has been a striking reduction in the degree of mitral regurgitation and in the L.A.P.

to penicillin. At 21 years of age she married and became pregnant; Cæsarean section was performed and she was sterilized. Thereafter she remained asymptomatic until December, 1958, when she developed a sore throat followed by joint pains, and a month later atrial fibrillation became established. Her symptoms progressively increased until she was almost completely disabled with congestive cardiac failure requiring hospitalization. Her response to diuretic and digitalis therapy was satisfactory, but her disability remained severe. Atrial fibrillation was present with a normal blood-pressure and jugular venous pressure. The apex was in the sixth space displaced outwards with an overfilled thrusting pulsation characteristic of severe mitral regurgitation. This was associated with a systolic thrill and a grade 4/6 pansystolic murmur, a loud third heart-sound, and a short, grade 2/4 mid-diastolic murmur (*Fig.* 777). There was no opening or closing snap of the mitral valve. Electrocardiography showed digitalis effect presumably on left ventricular hypertrophy and the radiograph (*Fig.* 778) showed considerable left ventricular enlargement with aneurysmal enlargement of the left atrium, but no valve calcium.

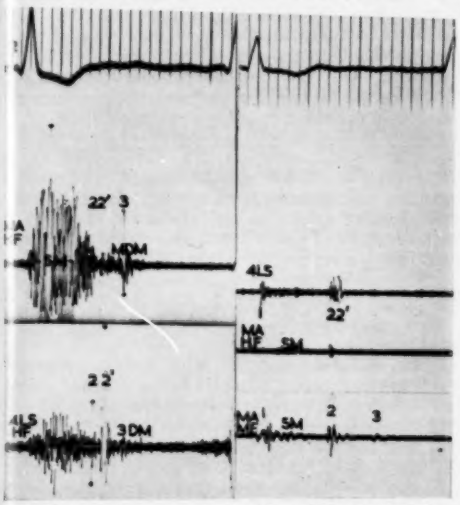
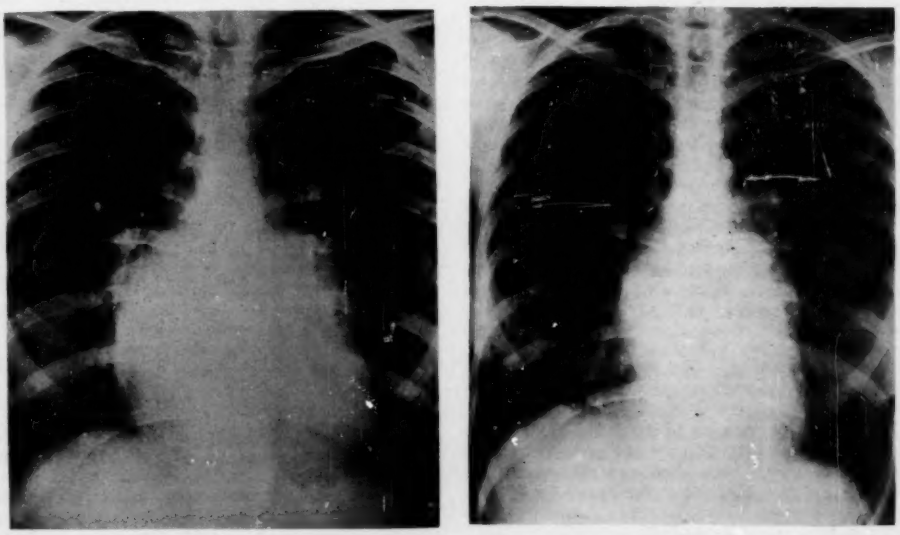


FIG. 777.—Prior to operation (*Case* 3) the phonocardiographic tracing at the mitral area (M.A.) shows a loud pansystolic murmur of mitral incompetence, followed by a mid-diastolic murmur and a loud third heart-sound. Splitting of the second heart-sound is wide (0·04 sec.) and the pulmonary component is considerably accentuated, being recorded at the apex. Three weeks after operation the apical systolic murmur has disappeared. The third heart-sound occurs later and is reduced in intensity and frequency, being recorded on medium frequency (M.F.) at the apex only. Splitting of the second sound is narrower and the pulmonary component is no longer accentuated. The first heart-sound has emerged. The lowest tracings have not been synchronously recorded, but have been superimposed from cycles of equal length. Atrial fibrillation is present.

At operation in September, 1959, with cardiac by-pass, pure mitral regurgitation was found. The mitral annulus was dilated and separation of the posterolateral cusp from the anteromedial cusp was found. An annuloplasty, as described above, was performed, approximating the two cusps, especially in the area of the medial commissure. Regurgitation was thereby corrected, and the anteromedial cusp could swing across and close up against the shortened posterolateral cusp. The striking relief of the incompetence is shown in *Figs.* 778, 779.

Within a fortnight of operation, the atrial fibrillation was corrected with quinidine, and the patient was soon discharged on a maintenance dose of quinidine and digitalis. Progressive improvement ensued. Radiography within 3 months of surgery showed a remarkable improvement (*Fig.* 778). However, in October, five days after stopping quinidine, atrial fibrillation recurred and correction with quinidine was again necessary. By November she was virtually asymptomatic and on maintenance quinidine only. The apex was now slightly displaced, with a marked decrease in the left ventricular thrust. A grade 3/6 systolic murmur was present, but



A B

FIG. 778.—A, The pre-operative radiograph (*Case* 3) shows considerable left atrial, left ventricular, and right atrial enlargement. B, Within 3 months of operation a striking reduction in size of all chambers has occurred.

with no third heart-sound or diastolic murmur. Sixteen months after operation she was virtually symptom-free.

SUMMARY

It is clear that the surgical correction of mitral regurgitation must be performed under direct vision, the exact technique employed depending upon the pathological anatomy of the diseased valve as visualized at open operation.

For practical purposes, closure of the normal mitral valve depends on: firstly, intact leaflets; secondly, mobility of the anteromedial leaflet; and lastly, a buttress against which this leaflet can close. It further seems logical that for the correction of mitral regurgitation, the surgeon must aim at providing these three factors.

Mitral regurgitation due to a cleft or perforation in one or another of the leaflets is easily corrected by the suturing of these defects. If, however, a valve insufficiency is due to the inability of a partly immobilized anteromedial leaflet to meet the posterolateral buttress, then the surgeon must aim at providing as much mobility as possible to the anteromedial leaflet, and, if necessary, also providing the posterolateral shelf. The various steps in achieving this are discussed. The treatment of those cases where gross deficiency of leaflet tissue exists is at present unsatisfactory, and the complete correction of this type of lesion awaits the development of a mobile prosthesis valve. A valve of this kind is being experimentally tested and will be the subject of a later discussion.

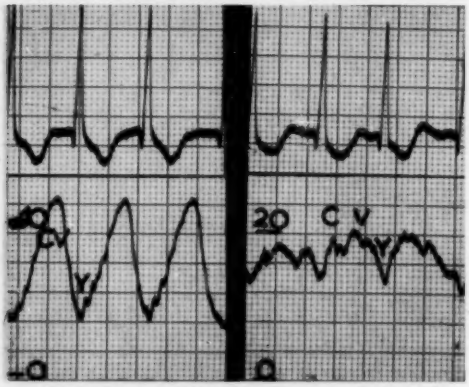


FIG. 779.—Tracing (*Case* 3) to show the dominant CV wave of gross regurgitation with no evidence of stenosis. After operation there has been a marked drop in L.A.P. with complete disappearance of mitral regurgitation.

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TUBERCULOUS EPIDIDYMITIS

A REVIEW OF 170 PATIENTS

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In the course of reviewing the results of treating 129 patients with tuberculous epididymitis at Wrightington Hospital during the last ten years, we felt that it would be of interest to compare present-day methods with an earlier series of 41 cases reported by one of us (J. C. R.) in 1948. In considering tuberculous epididymitis three main problems present themselves:—

1. Is it possible to define the precise route of infection to the epididymis?

2. Is tuberculous epididymitis becoming less frequent?

3. Is there a trend towards a more conservative approach in the treatment, similar to the tendency in renal lesions?

The uncertainty as to the precise route of infection to the epididymis led us to attempt to re-assess the three theories which have been postulated.

THE ROUTE OF INFECTION

1. Via the Lumen of the Vas.—This theory is widely held and envisages that tuberculosis of the epididymis is usually preceded by tuberculosis of the prostate or kidney. It is thought that tubercle bacilli reach the epididymis from the urine by migrating up the vas. That the retrovasal migration of bacteria may occur under certain conditions cannot be denied and evidence in support of this has repeatedly been observed. The acute epididymitis accompanying prostatic obstruction or urethral instrumentation would appear to be due to migration of organisms up the vas. Similarly many authors have observed a retrovasal flow of urine; Perves and Dubergey (1939) demonstrated contrast media in the ejaculatory ducts after urethrography. O'Connor (1935) quotes two patients in whom silver was found in the lumen of the vas after silver-nitrate irrigation of the bladder, whilst Ainsworth-Davies (1944), Hanley (1945), Meade (1947), and White and Berry (1956) have observed urinary fistulæ through the stump of the vas. Finally, stress epididymitis can only be explained by the passage of urine up the vas.

In practically all these examples, however, the retrovasal flow only occurs in the presence of some abnormality of the posterior urethra, produced by mechanical or inflammatory means which favour vasal stasis, and, therefore, retrovasal infection or urinary flow. Such abnormal conditions are now rarely found in genito-urinary tuberculosis owing to the changed pathological pattern since the advent of chemotherapy. In earlier days it can be appreciated that involvement of the prostate, whether from the blood-stream or from infected urine, could, through progression of the disease, produce a large cavity which ultimately connected with the prostatic ducts. Furthermore, before the present drugs were available Borthwick (1959) tied the contralateral vas when performing unilateral epididymectomy and in none of his cases did epididymitis develop; his numbers, however, were too small to be significant.

If, as stated above, the prostate is infected by a blood-borne organism, the lesion in the parenchyma, because of the powerful chemotherapeutic agents now available, is extremely unlikely to progress sufficiently to involve the prostatic ducts and to cause the large prostatic cavities so common prior to 1948.

Finally, in attempting to isolate tubercle bacilli from the urine, it is accepted that the organisms are present in very small numbers. This suggests that the minute amount of urine which could flow or be forced up the vas, even were there some gross abnormality of the posterior urethra to promote such a flow, would be most unlikely to contain many tubercle bacilli. In view of these facts we suggest that epididymitis is now rarely caused by tubercle bacilli passing up the vas.

2. Via the Lymphatics.—Supporters of this theory suggest that the spread of tubercle bacilli to the epididymis takes place from the urine, or the focus in the prostate, or the seminal vesicles via the lymphatics. This would necessitate a journey by the tubercle bacilli to the external iliac and pre-aortic lymph-glands and thence against the lymph-flow to the epididymis. This seems an unlikely route and should result in clinical tuberculosis of the lymphatic glands, which, in our experience, has not been observed.

3. Via the Blood-stream.—It is commonly accepted that renal tuberculosis is produced by a blood-borne infection of the kidneys. This is irrefutable proof that at some time a shower of tubercle bacilli must have been present in the general circulation. It is inevitable, then, that elements of this shower will be carried by the blood-stream to all parts of the body, including kidneys and epididymides. The result of the arrival of the tubercle bacilli in any tissue will depend upon the resistance of that tissue to the growth and multiplication of the bacilli and the type and virulence of the infecting tubercle bacilli. Such structures as muscle, testes, skin, and female genitalia are relatively immune; others, such as kidney, epididymides, bone, and joints, are relatively susceptible and, therefore, frequently invaded.

In accepting this concept, on every occasion that tubercle bacilli reach the kidney it is probable that some organisms will also reach the testes, epididymides, and prostate. The testicular tissue, being resistant, does not allow the development of tuberculous foci, but the epididymis, being susceptible, readily encourages the multiplication of bacteria. Whether one or both epididymides eventually show clinical evidence of tuberculosis and the time at which such evidence appears will depend on the number of tubercle bacilli and the resistance put up by the tissue.

This brings tuberculous epididymitis exactly into line with renal tuberculosis and we believe that, as in renal tuberculosis, a careful study of the contralateral epididymis in cases of unilateral disease would reveal microscopical tuberculous foci in the majority of cases.

Since the blood-borne theory is logical, fits the observed facts, and allows epididymal tuberculosis to conform with other secondary manifestations of tuberculosis, we believe that it is responsible for the majority of infections.

With regard to the second two questions, the following study attempts to give satisfactory answers. One thing, however, is certain—that in the recent decade the whole outlook in renal and epididymal tuberculosis has changed; this is only to be expected as the two conditions are closely associated and a great number of patients with tuberculous epididymitis have either an overt renal lesion or tuberculous bacilluria secondary to a small Medlar's focus.

COMBINED GENITAL AND URINARY LESIONS

Wells (1943) considers that tuberculosis of the epididymis, even without an overt renal lesion, indicates that the kidney is involved, basing this opinion on a series of 55 patients with epididymitis, of whom 30 subsequently developed renal tuberculosis. Our experience supports this view; in our earlier group 40 per cent had associated clinical renal tuberculosis and others had tubercle bacilli in the urine. In the later series of 129 cases 65 per cent were found to have a positive urine at some stage. A difficulty which arises here is that the tubercle bacilli may come from the prostate and seminal vesicles, and in one such patient it was possible to find numerous tubercle bacilli in a specimen of urine passed immediately after prostatic massage.

The question has often been asked why it is that concomitant genital and renal tuberculosis is so common in the male and so uncommon in the female. In our series at Wrightington the latter was only observed in 1 per cent of cases. Perhaps the different embryological origin of the structures may be a possible explanation. The epididymis (which has a very liberal blood-supply) arises from the mesonephric duct, whereas in the female this structure is reduced to three remnants—(*a*) the duct of the epoophoron, (*b*) the vesiculæ appendices, and (*c*) the remnants of Gaertner, situated in the broad ligament. The blood-supply of these three remnants arises from the utero-ovarian anastomosis and it is, therefore, most unlikely that they would ever be affected by tubercle bacilli.

MATERIAL

In the earlier group 39 epididymectomies and 2 orchidectomies were performed and in the later 154 epididymectomies and 8 orchidectomies. Twelve were treated by chemotherapy alone.

Clinical Features.—For the purposes of comparison, the patients were divided into two groups, i.e., the 'earlier' or pre-chemotherapeutic series, and the 'later' series, treated by modern chemotherapeutic methods.

Very little difference was found in the two groups as far as clinical features were concerned.

Age.—As might be expected, the disease develops during the period of the greatest sexual activity and the majority of patients in both groups are included in the two decades between 20 and 40 years (*Table I*).

Table I.—Age Incidence

Ages	*Number of Cases* 1948 *series*	*Later series*
Under 20 years	7	6
20–30 years	11	43
30–40 years	13	32
40–50 years	6	22
50–60 years	3	20
60 years +	1	6
Totals	41	129

Previous History of Tuberculosis.—A previous history of tuberculosis was given in 70 per cent of the earlier series and 76 per cent of the later group. The majority had suffered from pulmonary tuberculosis; although there were a few cases of previous bone and joint involvement a previous history of renal tuberculosis was rarely obtained.

Presenting Symptoms (*Table II*).—It appears that patients now seek medical help earlier, as the great majority in the later series were referred to the centre within six months of the onset of symptoms, whereas the similar time-period was seven months or over in the pre-chemotherapeutic group.

Table II.—Presenting Symptoms

1948 *Series*		*Later Series*	
Scrotal swelling	26	Scrotal swelling	108
Swelling and pain	15	Swelling and pain	9
(Many complaining of swelling also had a sinus)		Pain	7
		Swelling and discharge	2
		Scrotal sinus	3

It is obvious that pain is infrequent and that the acute fulminating variety is extremely rare. Trauma was only recorded on 6 occasions in the later series.

Physical Signs.—The two sides were equally affected in both series and the condition was bilateral in 34 per cent. Approximately half the patients in both groups had an abscess or sinus when first seen. *Table III* indicates the part or parts of the epididymis involved, the figures emphasizing the fact that when the disease is localized, the condition is usually found in the globus minor. It was surprising that, even in the later series, a great many patients (over a hundred) had widespread disease involving all three parts of the epididymis. The small percentage of secondary hydrocele, i.e., 5 per cent, is considerably less than that recorded in most text-books.

The vas was thickened in 74 per cent of the earlier cases and in approximately 50 per cent of the more recent group, but beading was exceptional. It is clear that the pathology of the vas has a fundamental bearing on treatment and it is obviously wrong to bury the stump of the vas in the tissues if it is infected.

Table III.—Part of Epididymis Involved

	Later Series
Globus minor	33
Globus major	8
Body	0
All three	113
Globus major and globus minor	9
Globus major and body	2
Globus minor and body	9
Hydrocele	7

Sexual Aspects.—Veenema and Lattimer (195), in a review of 60 patients with genital tuberculosis, found that fertility was considerably diminished and

that sperm counts and motility were generally reduced. The[r] considered that this was caused partly by bloc[k]age of the vas and partly by secondary atrophy. In [2] patients out of 35 a positive culture for tubercle bac[illi] was obtained from the seminal fluid.

Treatment.—It has always been maintained by us that the treatment of tuberculous epididymitis is merely an incident in the treatment of genito-urinary tuberculosis, although the genital lesion may be the most prominent clinical feature. In other words, tuberculous epididymitis is a local manifestation of a constitutional disease. Having established this fundamental concept, it follows that all patients with tuberculous epididymitis must receive the full course of treatment advocated by us in previous communications (Ross, Gow, and Hill, 1951, 1953, 195[5]). This involves six months' intensive chemotherapy in a sanatorium followed by domiciliary courses of treatment spreading over a period of two years or more. Only by so doing can we hope to prevent the disease from developing in some other situation in the body. If operation is necessary we believe strongly that the only permissible procedure in the vast majority of cases is epididymectomy (*see below*). It used to be said that it was a sound principle to remove any tuberculous focus which could be eliminated easily. Since the advent of chemotherapy, however, this does not apply to tuberculous epididymitis and it now appears that removal of the epididymis is necessary only when the condition fails to respond to an adequate course of chemotherapy. Comparison of the two groups is particularly interesting in this respect. In the group dealt with before 1948 practically all patients required epididymectomy, whereas, since the advent of chemotherapy 12 patients with epididymitis did not require operation, the epididymis either returning to normal or becoming a fibrous cord. Sinuses healed well and many abscesses resolved satisfactorily, although sometimes aspiration was necessary. We have been impressed by the fact that each year fewer patients appear to need operative treatment, this point being well shown on *Table IV*.

Table IV.—Epididymectomy by Years

Year	*Later Series No. of Cases under Treatment*
1948	11
1949	17
1950	14
1951	14
1952	13
1953	11
1954	17
1955	13
1956	9
1957	5
1958	5

At the present time we consider the following complications indicate operation:—

1. Gross thickening of the vas and cord.
2. A large caseating abscess.
3. Failure to respond to treatment.

There is undoubtedly an optimum time for operation and, as the treatment is prolonged, there is no need to interfere before assessment of the response to therapy. In our experience it is particularly important to avoid operating on a confluent inflammatory mass in which the epididymis and the body of the testis are indistinguishable. If patience is practised in this type of case the mass gradually resolves, so that if operation ultimately becomes necessary it is possible to dissect the epididymis from the body of the testis, which is carefully preserved.

A word about alternative operative procedures. Orchidectomy is mentioned only to be condemned; it is unnecessary except in a few cases, it is mutilating, and ends up in castration in the frequent bilateral case. The radical operation, involving the removal of the epididymis and the vas deferens by the inguinal route and the removal of the seminal vesicles and the prostate *en bloc* through a perineal incision, as advocated by Young and Davis (1926) is fundamentally unsound as large tissue spaces are laid open to potential infection. Similarly, Loughnane's operation (1945), removing the testis, vas, and seminal vesicle on the same side, is open to the same objection.

Some authorities advocate division and ligation of the contralateral vas when carrying out an epididymectomy, but we have always opposed this view on the grounds that it provides against a complication which may never happen at the great cost of rendering the patient permanently sterile. Experience has confirmed us in the opinion and in the course of time other contra-indications to the procedure have appeared and include:—

1. Chemotherapy, provided it is adequate, has made subsequent epididymitis on the opposite side unlikely (*see Table V*); a prolonged follow-up extending over a period of over 10 years has confirmed this.

2. Division and ligature of the vas does not invariably prevent epididymitis after prostatectomy.

3. Several of our patients fathered children after unilateral epididymectomy.

Table V.—Contralateral Epididymitis developing after Epididymectomy

1948 Series	*Later Series*
Already bilateral in half the patients when first seen	22 cases:— 18 prior to 1952 4 since 1952 0 since 1955

The technique of epididymectomy has been previously described by one of us (J. C. R., 1948) and it is unnecessary to describe the operation in detail here. The operation is carried out through a scrotal incision which includes an ellipse of skin if a sinus is present. The two fundamental principles are the preservation of the blood-supply of the body and the exteriorization of the stump of the vas deferens, which is brought out through a stab incision over the external ring. It has been pointed out above that the vas is usually infected, and it is obviously wrong to allow the stump to retract back into the properitoneal tissues and to cause an abscess or sinus. The body of the testis is resistant to tuberculosis and, provided the dissection is carried out with care and the blood-supply preserved, will survive in spite of extensive disease and even abscess formation between it and the epididymis. Hæmostasis is important and we have found that the post-operative support and gentle pressure afforded by a St. Andrew's cross bandage prevents hæmatoma formation. First-intention healing of the scrotal wound results, the small half-inch vasal stump sloughing off in the course of 10 days and leaving a small depressed scar.

Results of Treatment.—In studying these patients every effort has been made to achieve as complete a follow-up as possible and all but 2 of the patients now living have been located and examined, with the help of physicians, surgeons, general practitioners, and tuberculosis authorities in many different parts of the country.

In the earlier group 3 patients died from general tuberculosis whilst in hospital, the remaining 38 being ultimately discharged satisfactorily healed. Of these 38 patients at least 5 succumbed later to tuberculosis affecting other organs. Among the survivors 16 apparently 'normal' testes were found, the body in each case being well preserved and showing no sign of atrophy.

In the 129 patients treated in the chemotherapeutic era there were 32 deaths, none during the immediate post-operative period. It should be noted that many of these occurred in the earlier part of the decade and few patients have died in recent years.

Table VI.—DEATHS

Year	*No. of Deaths*
1948	1
1949	6
1950	3
1951	6
1952	—
1953	1
1954	4
1955	5
1956	2
1957	4
1958	0

Of the 11 deaths since 1954 only 3 have been due to tuberculosis, the first chronic pulmonary disease, the second uræmia and the third amyloid disease due to longstanding tuberculosis of the spine.

A positive urine following epididymectomy was the next factor studied and *Table VII* shows the results.

Table VII.—POSITIVE URINE AFTER EPIDIDYMECTOMY

1948 Series	*Later Series*
16 patients had an associated renal lesion with a positive urine	41 patients:—
	23 with associated renal lesion
	18 with no renal lesion

The urine of all patients in the later series was sterilized by a further prolonged course of chemotherapy. The results of epididymectomy have on the whole been satisfactory, the morbidity being minimal and post-operative mortality nil. Atrophy of the testis has occurred in 11 per cent of patients, rather higher than usually quoted in text-books, perhaps due to the fact that epididymectomy has been attempted on every possible occasion, even when it was realized that the blood-supply to the testis was precarious.

It is salutary and gratifying to record that how many cases of tuberculous epididymitis heal without surgical intervention, but in such cases the remote possibility of carcinoma of the epididymis should be appreciated. One such case has been encountered in this series (Gow, 1957).

We believe that a swelling of the epididymis which has not decreased in size after at least four months' treatment should be explored, as most tuberculous lesions will improve in that time.

SUMMARY

1. A group of 129 patients with tuberculous epididymitis is compared with an earlier series of 41 dealt with before chemotherapy was available.

2. The possible routes of infection are discussed.

3. The close association between renal and genital tuberculosis is emphasized.

4. Thickening of the vas deferens was frequent but beading and secondary hydrocele were rarely seen.

5. Patients with epididymitis require a full course of chemotherapy in a sanatorium in the same way as those with renal tuberculosis.

6. With modern methods of treatment it was found that epididymectomy was needed much less frequently.

7. It was pointed out that epididymectomy is the only permissible operation and that, provided the vas is exteriorized and the blood-supply of the body preserved, the results are eminently satisfactory.

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HÜRTHLE-CELL CARCINOMA OF THE THYROID

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Hürthle cells occur in normal thyroid glands and in thyroids showing various disease states, e.g., Hashimoto's disease, colloid goitre, thyrotoxicosis, and infiltration by tumour growth. Neoplastic Hürthle cells occur in adenomas and adenocarcinomas of the thyroid and may comprise a considerable proportion of the tumour, but it is difficult to assess to what extent, if any, they influence the course of the disease, favourably or otherwise. There is some evidence that pure Hürthle-cell tumours may be of relatively low-grade malignancy, although as yet too little is known of their growth characteristics to be certain of this.

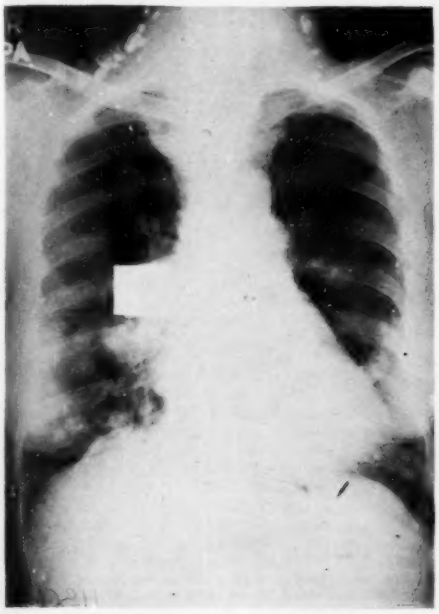

FIG. 780.—Radiograph of chest. Multiple secondary tumour deposits in both lungs; calcified tuberculous glands in the neck.

The following case of Hürthle-cell carcinoma is of particular interest in that no treatment for the thyroid lesion was given at any time during the course of the illness, and in consequence it reveals something of the natural history of the disease process.

CASE REPORT

From the age of 30 years until her death at 62 years, M. H., a married woman, had a goitre which varied in size from time to time but never disappeared. At about 48 years of age she developed a brassy cough, and at 52 began to have dyspnœa on exertion, but there was no dysphagia or symptoms of thyrotoxicosis. At 53 years, the chest was examined radiologically (April, 1949) and multiple nodules of secondary tumour were observed in the lungs.

Her other history was not relevant to the thyroid condition. In infancy she had multiple abscesses in the neck, and from early on in childhood it had been noted that her right leg was slightly shorter than the left. At the age of 46 years, she had menorrhagia, which was treated successfully by a castration dose of X rays to the ovaries.

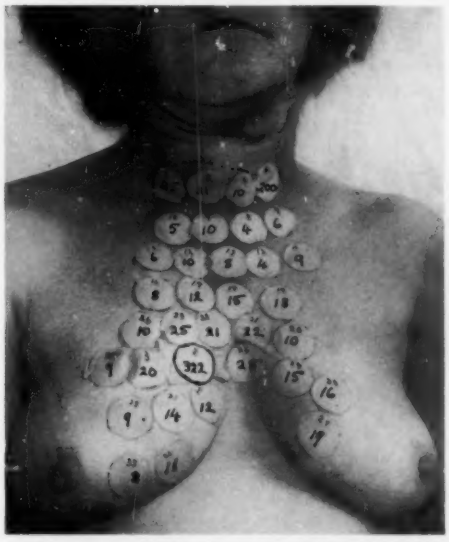

FIG. 781.—The tumour in the thyroid gland, outlined by interrupted lines, shows no uptake of radio-active iodine, as the counts per minute are small. Disk 2, outlined by a continuous line, is over the pulmonary metastasis where there was a high uptake of radio-active iodine, indicating the presence of a functioning thyroid metastasis.

Eight months later she had three massive hæmatemeses necessitating blood transfusion (group O blood). At that time there was no abnormality of the cardiovascular or respiratory systems. The liver and spleen were enlarged, and there was severe anæmia—Hb 20 per cent; R.B.C.s 1,000,000 per cu.mm.; W.B.C.s 3100 per cu.mm.; platelets 108,000 per cu.mm. She was diagnosed as Banti's syndrome. Her anæmia responded favourably to iron therapy.

She was first seen by us in May, 1950, when the pulmonary metastases had been present for over a year.

ON EXAMINATION.—She was well nourished, not anæmic, and her general condition was good. In the right lobe of the thyroid there was a swelling which resembled a benign adenoma. Numerous, small, firm, shotty glands were present in both sides of the neck. Clinically there

were no abnormal features in the central nervous system and cardiovascular system. Scattered sonorous rhonchi were heard in the chest. On laryngoscopy the larynx was tilted but the vocal cords moved normally. The tongue was smooth. The spleen was enlarged.

INVESTIGATIONS.—A repeat radiograph showed multiple secondary deposits in the chest. There was a partly calcified mass in the right lobe of the thyroid gland, with marked tracheal displacement. Old calcified glands of apparently tuberculous origin were present in both sides of the neck (*Fig.* 780). Hb 92 per cent, 13·6 g. per cent. W.B.C.s 3000 per cu.mm.; neutrophils 1450 per cu.mm.; lymphocytes 1400 per cu.mm.; monocytes 150 per cu.mm. The blood film showed no abnormality in the red corpuscles. Basal metabolic rate plus 4 per cent. Tracer studies with

FIG. 782.—Posterior view of the tumour occupying the right lobe of the thyroid. (× ⅓.)

I^{131} showed a normal uptake by the thyroid itself but none by the thyroid tumour. One pulmonary metastasis (*Fig.* 781) showed a high take-up, and it was concluded that the pulmonary metastases originated from a carcinoma of the thyroid.

The patient was given no form of treatment, but was followed-up at yearly intervals for the next six years. During that time she felt better, but still had a brassy unproductive cough and had dyspnœa on exertion. The thyroid swelling felt smaller, but some hard glands developed in the right supraclavicular triangle.

At the age of 62 years she was readmitted in coma with cyanosis, moist respirations, and laryngeal stridor. There was massive œdema of the arms, legs, abdominal wall, and face. She died a few hours later.

NECROPSY FINDINGS.—The significant findings other than those relevant to the Hürthle-cell tumour and its metastases were: pallor of the skin and mucosæ; no evidence of wasting; well-marked œdema of the legs and upper arms; effusions of straw-coloured fluid in the peritoneal cavity (590 ml.) and in both pleural cavities (approx. 750 ml.); fibrino-sanguineous fluid in the pericardium (approx. 300 ml.); extensive fibrinous pericarditis; hypertrophy of the right and left ventricles (heart weight 449 g.); fibrous atheromatous plaques throughout the aorta; fibro-congestive splenomegaly (600 g.), chronic perisplenitis; three old splenic infarctions; varicosity of the splenic vein and thrombosis at its junction with the portal vein, the thrombus partially occluding both vessels; venous congestion of the small intestine, especially the ileum; gross arteriosclerosis and aneurysmal dilatations of the splenic artery, and also of

FIG. 783.—Section of right lung showing multiple tumour metastases and also a recent hæmorrhagic infarction. (× ⅓.)

the short gastric arteries; numerous varicosities of veins around the cardia and in the lower end of the œsophagus; normal-sized liver with small areas of fibrosis, but no secondary tumour deposits; active red-marrow throughout the right femur associated with rarefaction and thinning of the cancellous and compact bone; Legg-Calve-Perthes disease of the right femoral head.

Thyroid.—The right lobe was enlarged, measuring 8 cm. in length, 4·5 cm. in width, and 5·5 cm. in depth. On section, it was entirely occupied by an encapsulated tumour of nodular pattern, the nodules varying from 0·5 cm. to 2·5 cm. in diameter and consisting of soft tissue of fawn or brown colour; the fibrous septa between the nodules contained several extremely calcified areas. At the upper and lower poles there were two or three distended, thrombosed vessels (*Fig.* 782). The left lobe and the isthmus were normal.

Lymph-nodes.—Calcified healed tuberculous lymph-nodes were present in both sides of the neck. The paratracheal nodes, especially the left, were enlarged up to 2·5 cm. diameter. On section they contained nodules of tumour tissue similar in appearance to that in the right lobe of the thyroid. In the superior mediastinum there was

slight side-to-side compression of the trachea due to a mass (5 cm. wide × 5 cm. deep) of enlarged lymph-nodes invaded by tumour. At the bifurcation of the trachea and at the lung roots, the lymph-nodes were enlarged and largely replaced by tumour growth.

Lungs.—Left lung: the upper lobe showed an area of infarction involving the anterolateral and anteromedial segments, and in the lower part of the lobe several small, rounded, fawn-coloured tumour nodules; scattered throughout the lower lobe there were about ten tumour nodules varying from 1 cm. to 3 cm. in diameter. Right lung: in the upper lobe there was infarction of the anterolateral segment, and in the lower half of the lobe multiple tumour nodules up to 3 cm. in diameter (*Fig.* 783); the middle lobe contained a few small nodules of tumour; the lower lobe showed an area of infarction in the posterior basic segment and elsewhere scattered tumour nodules up to 3 cm. in diameter. Small thrombosed vessels compressed by tumour nodules were noted in relation to each of the infarcted areas. The main pulmonary arteries, although intimately surrounded by tumour-invaded lymph-nodes, contained no thrombi. There were no fibrous adhesions over the pleura, and no tumour deposits in the pleural or pericardial cavities.

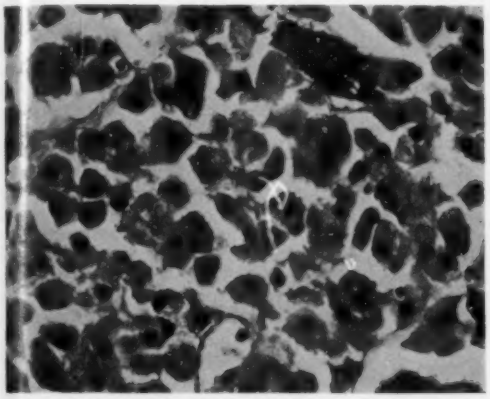

FIG. 784.—Neoplastic Hürthle cells with deeply eosinophilic, finely granular cytoplasm and dark-staining nuclei; also a focus of calcification which is partly intracellular. H. and E. (× 510.)

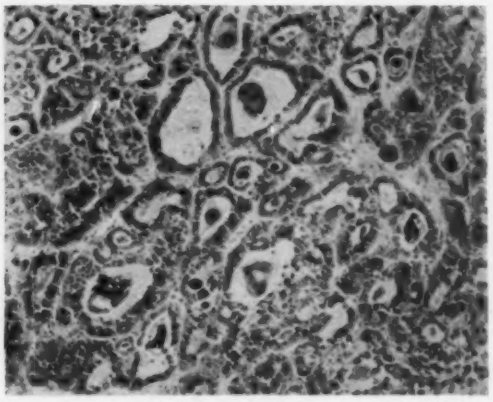

FIG. 785.—Right lung metastasis from the region of high I^{131} uptake. Well-formed acini with rather poorly-stained colloid. H. and E. (× 70.)

MICROSCOPY.—

Thyroid.—Histological examination of the right lobe of the thyroid revealed a very cellular tumour consisting of rather large, deeply eosinophilic, epithelial cells with finely granular cytoplasm. The cells were of varied shape, some rounded, others rectangular or polyhedral. The nuclei were dark-staining, but mitotic activity was not observed (*Fig.* 784). In general the cells formed small irregular clumps or larger masses separated by thin vascular fibrous-tissue trabeculæ; intercellular spaces and spaces between cells and their basement membranes were conspicuous features. In some parts of the tumour a broad trabecular arrangement was apparent, and in a few areas there were small acinar formations, some of which contained poorly-stained colloid. The tumour was intersected by fibrous-tissue septa and was surrounded by a fibrous capsule of variable width. There were numerous small foci of calcification both in the broader septa and in the stroma of the cellular parts of the tumour, especially where hyaline change had occurred. At many sites the capsule was infiltrated by small clumps of tumour tissue. Several blood-vessels were similarly infiltrated, but tumour cells were not demonstrable in the lumen.

Nearly 80 per cent of the tumour in the right lobe was infarcted, most of the infarcts being about 1–3 days old. Many of the larger veins contained thrombi, also of recent formation, but one or two vessels were completely occluded by old hyaline, collagenous tissue. At the upper and lower poles in particular, there were some densely fibrosed areas containing large calcareous deposits. In addition, amongst the infarcted cellular parts of the tumour, there were several places where the stroma consisted of a loose hyalinized collagenous framework enclosing spaces, some of which were empty, while others included calcium deposits, foam cells, or very small clusters of deeply eosinophilic neoplastic Hürthle cells.

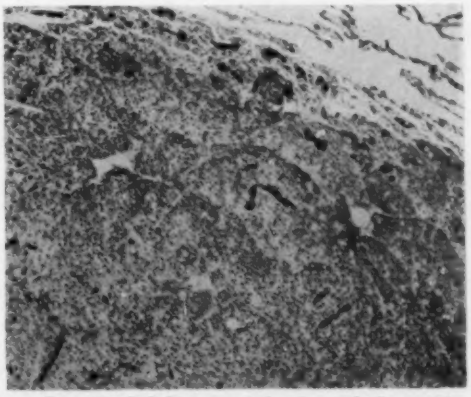

FIG. 786.—Left lung metastasis. A completely necrotic tumour nodule showing a 'tidal zone' appearance. H. and E. (× 32.)

Two normal parathyroid glands were identified histologically in the connective-tissue capsule of the right lobe. Apart from compression and atrophy of acini in the isthmus immediately adjacent to the tumour, the remainder of the thyroid was normal.

Lymph-nodes.—The metastases in the cervical, paratracheal, and mediastinal lymph-nodes all showed features similar to those in the primary growth, viz., necrosis, recent infarction, and thrombosis of blood-vessels, fibrous

encapsulation and fibrous septa, foci of hyalinization of the stroma and of calcification. A few small hæmorrhages and occasional small aggregates of hæmosiderin-filled macrophages were also present.

Lungs.—Fifteen tumour nodules from each lung were examined. The histological pattern of the tumour was unchanged except in two or three nodules from the anterior middle and dorsal segments of the right lung where the follicular pattern was more conspicuous than elsewhere. One large nodule in particular showed a high proportion of well-formed acini with rather poorly-stained colloid (*Fig.* 785). In general there was much necrosis and infarction, mostly recent in origin (about 1–3 days) but occasionally much older (months or years). The necrosis was mainly in the central parts of the nodules, but sometimes occurred around the periphery. Some nodules were completely necrotic and showed a 'tidal zone' appearance (*Fig.* 786). Several of the pulmonary metastases had a thick fibrous capsule and broad hyalinized fibrous septa, and in many there were frequent small foci of calcification (*Figs.* 787–789). This calcification affected not only the connective-tissue stroma but also the cellular parts of the tumour, certain tumour cells showing calcium deposits within the cytoplasm, and some of the smaller acini containing calcified colloid material in the lumen.

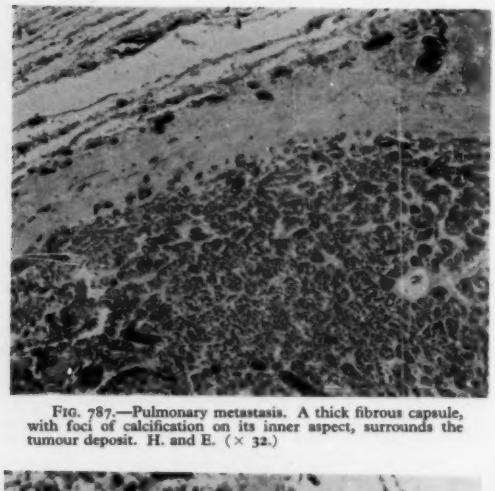

FIG. 787.—Pulmonary metastasis. A thick fibrous capsule, with foci of calcification on its inner aspect, surrounds the tumour deposit. H. and E. (× 32.)

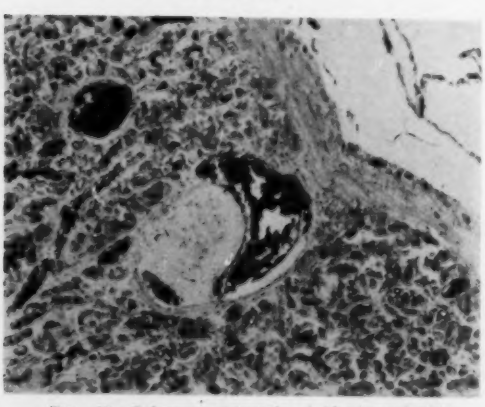

FIG. 788.—Pulmonary metastasis. A blood-vessel within the tumour deposit shows an old thrombus partially recanalized. H. and E. (× 70.)

Other Organs.—The liver showed chronic venous congestion, and in the lateral part of the left lobe this was accompanied by recent necrosis of liver lobules and by thrombosis of the hepatic artery in one or two portal tracts. The spleen showed congestion of the sinusoids, fibrosis of the stroma, small areas of perivascular calcinosiderosis in the trabeculæ, and hyalinized fibrous thickening of the capsule. There was marked passive congestion of the submucosal vessels of the ileum, and in the related mesentery some of the smaller branches of the superior mesenteric artery were thrombosed. The kidneys showed a minor degree of subcapsular focal sclerosis and in the medulla a few foci of interstitial calcification. The pituitary, adrenals, and ovaries were normal. In the lower end of the right femur, the bone-marrow showed a normoblastic hyperplasia; there were also one or two areas of recent infarction or necrosis of the soft cellular marrow. The articular part of the head of the right femur showed pronounced osteoarthritic changes; in the cancellous part there were numerous areas of necrosis and fragmentation of bony trabeculæ; some of these areas showed no surrounding reaction, others showed a rather loose fibrous tissue containing small osteoid deposits, whilst in one or two such areas there was a chronic inflammatory infiltration including lymphocytes and plasma cells. Similar changes were present in sections from the lower thoracic vertebræ.

DISCUSSION

On the observations of several authors there is evidence to the effect that Hürthle-cell carcinomas are slowly growing tumours (Morrow, 1945; Chesky, Dreese, and Hellwig, 1951; Goldenberg, 1953; and Gardner, 1955). Frazell and Foote (1949), however, reported that in a series of 40 cases of Hürthle-cell cancer only one-third of the patients were alive and free of disease 5 years after their initial treatment. Their results would suggest that this tumour is no less malignant than other types of thyroid cancer. Such discrepancy over the degree of malignancy may be partly explained by differences in the histological classification of those tumours, in particular by differences in the accepted criteria of malignancy. In establishing the malignancy of a tumour of the thyroid, one cannot rely entirely on histological criteria such

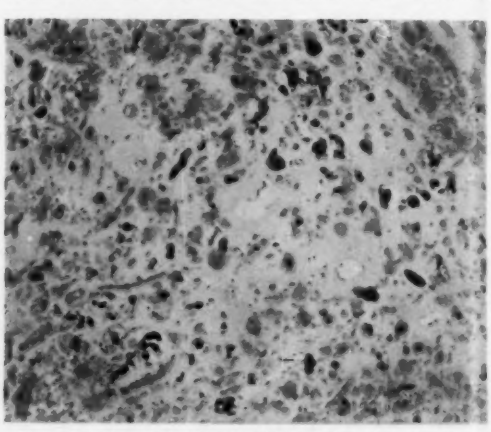

FIG. 789.—Pulmonary metastasis. Multiple foci of calcification and hyalinization of the stroma. H. and E. (× 55.)

as invasion of the capsule or invasion of vessels. Often, it is the late recurrent growth in the thyroid, or the development of glands in the neck or mediastinum or of metastases in the lungs or bones years later, that proves beyond doubt its malignant character. By study of such indisputably malignant cases, accurate information can be accumulated regarding the growth characteristics of the Hürthle-cell carcinoma.

In the case reported above, the malignant nature of the disease was first demonstrated radiologically 9 years prior to her death. How long the lung metastases had been present before this, one cannot accurately determine. Six months previously she had become dyspnœic on exertion, suggesting that pulmonary metastases, or at least mediastinal involvement, had been present at that time. Indeed, one could speculate that even several years earlier, the onset of a 'brassy' cough was due to mediastinal involvement. However, the established fact is that the pulmonary metastases had existed for a period of at least 9 years.

The initial recognition that the primary growth was located in the thyroid was based on tracer studies with radio-active iodine. One secondary nodule in the right lung showed high I^{131} activity. This suggested that the pulmonary metastases had originated from a carcinoma of thyroid, even although clinically the swelling in the thyroid was like a benign adenoma. The diagnosis was amply confirmed at post-mortem examination and in addition the lung secondaries in the area of high I^{131} activity were found to have a greater proportion of acini as well as larger acini than any other part of the tumour, primary or metastatic (*Fig.* 785).

The only treatment given to this patient was several years before the metastases appeared, and included a castration dose of radiation to the ovaries for menorrhagia, and iron and blood transfusion for her Banti's syndrome. The swelling was present in her neck at that time but it seems unlikely that such therapy could have modified the course of the disease in the thyroid. Although it was tempting to treat this patient with a thyroid-ablative dose of I^{131}, followed by a possible therapeutic dose, considered this inadvisable as the primary in the thyroid, and the majority of the pulmonary metastases, did not pick up I^{131}. If she had been treated in this way, she certainly would have developed myxœdema, with its own attendant morbidity and mortality. This may or may not have resulted in the tumour later taking up I^{131} and causing some regression of the malignant process. Even so, we have yet to see a similar case of Hürthle-cell carcinoma with proved secondaries in the lung treated with I^{131}, radiotherapy, surgery, or a combination of these methods, which has survived so long.

Histological investigation of this case revealed several features in keeping with a carcinoma of low-grade malignancy. For example, in spite of considerable variation in cell size and shape, there was no evidence of mitoses in the primary growth or in any of the secondary deposits in the lymph-nodes or lungs. The rather small, darkly-staining nuclei in certain parts of the tumour gave the impression of pycnotic degeneration rather than the nuclear hyperchromatism of a rapidly growing tumour. The presence of calcium deposits within the cytoplasm of some of the tumour cells indicated cellular degeneration. In addition the stroma of the tumour showed well-marked retrogressive changes such as fibrosis, hyalinization, and numerous foci of calcification. These changes are not uncommon in benign adenomas of the thyroid. When they are seen in pulmonary metastases, as in the above case, it is reasonable to assume that the tumour is one of relatively low-grade malignancy.

A histological feature of outstanding interest was the extensive infarction of the primary growth in the thyroid and of many of the secondaries in the lungs and in the paratracheal and mediastinal lymph-nodes. In the thyroid tumour, the infarcted areas and the associated vascular changes indicated vascular disturbances of different ages, mostly 1–3 days, but in some parts months or even years old. In the lungs certain secondary nodules consisting almost entirely of necrotic or infarcted tumour tissue showed what appeared to be tree-like 'rings of growth', as if repeated episodes of necrosis or infarction had occurred, and following each episode only the live peripheral zone had survived. Vascular thrombosis leading to infarction within the tumour growth and its secondaries may thus have partly, if not wholly, accounted for the very long survival. It is difficult to dissociate aetiologically the vascular thrombosis and infarction within the tumour from that occurring elsewhere in this case (viz., pulmonary, hepatic, portal, and splenic thromboses and possibly bone-marrow), particularly in the presence of congestive cardiac failure which may by itself predispose to intravascular thrombosis. It would be of interest therefore to know to what extent vascular thrombosis and infarction are found in Hürthle-cell carcinoma uncomplicated by other conditions associated with thrombus formation.

Review of the literature shows several cases of treated Hürthle-cell carcinoma with long survival after the development of lung secondaries. Haagensen (1931) reported one woman, 58 years old, who survived 6 years. Horn (1954) has recorded 2 such cases, one a woman of 80 years who was still alive 2½ years after the recognition of the lung secondaries, the other a woman of 60 years who developed secondaries in the lung about a year after an incomplete resection of the primary in the thyroid and who subsequently survived a further 9 years. Frazell and Duffy (1951) also refer to one remarkable case in which minimal doses of X rays to the chest resulted in complete disappearance of 'alleged' metastatic lesions; their patient remained well for 10 years and died from other causes. Our case, in surviving 9 years, appears to be the longest survived case reported of a Hürthle-cell carcinoma with proved pulmonary metastases. In general the life expectancy in other types of untreated thyroid cancer with spread to the lungs is very short, a matter of months. It may be that the above case is exceptional but at present it would tend to favour the viewpoint that the Hürthle-cell carcinoma is of relatively slow growth.

SUMMARY

A case of untreated Hürthle-cell carcinoma of the thyroid surviving 9 years with widespread, proven

pulmonary metastases is described. Radio-active iodine tracer studies were of diagnostic value in the initial location of the carcinoma in the thyroid. The histology of the tumour and its metastases showed features in keeping with a carcinoma of relatively slow growth.

Acknowledgements.—It is a pleasure to thank Dr. E. Wyn Jones for allowing us access to his patient; and Professor H. L. Sheehan for his helpful advice. We are indebted to Mr. D. J. Andrews for the photographs.

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SPINAL CORD COMPRESSION IN HODGKIN'S DISEASE

A REVIEW OF 10 CASES

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SINCE Murchison and Sanderson described the first case of involvement of the central nervous system by Hodgkin's disease in 1870 there have been a series of reports, notably by Ginsburg (1927), Verda (1944), Shenkin, Horn, and Grant (1945), and Vogel and Richland (1955), of cases of Hodgkin's disease presenting with spinal symptoms alone and no other manifestations of the disease. It may not, however, be sufficiently well realized that central nervous symptoms may be the only manifestation of the disease for several years or even a lifetime and we therefore felt that it would be valuable to review our own series of ten cases. These patients were admitted under the care of one of us (W. McK.), either to the National Hospital, Queen Square, or Atkinson Morley's Hospital, between 1937 and 1960. Nine of these cases were verified by laminectomy but in the remaining case the diagnosis of Hodgkin's disease had been proved by removal of a lymph-gland and the diagnosis was confirmed at autopsy.

Sex Incidence.—Of these 10 cases 6 were males and 4 females, an incidence ratio of 3: 2, a finding similar to that in generalized Hodgkin's disease in which 70 per cent of the patients affected are males (Jackson and Parker, 1947).

Age Incidence.—*Table I* reveals that in no case did the disease appear before the age of 40 and that 2 patients were in the eighth decade.

Table I.—AGE INCIDENCE

Age	*No. of Cases*
40–49 yr.	2
50–59 yr.	4
60–69 yr.	2
70–79 yr.	2

Incidence of Primary Spinal Involvement.—Of these 10 cases only 2 had other manifestations of the disease and had been diagnosed as suffering from Hodgkin's disease before spinal symptoms appeared. The other 8 cases presented primarily with symptoms and signs of spinal involvement. Of these 8 cases only 2 were subsequently found to have developed other evidence of the disease, one having shown enlargement of lymph-glands 3 months later and the other having multiple deposits which were found at autopsy.

Site of Lesion.—The site of the spinal lesion was cervical in 1, dorsal in 7, and lumbar in 2 cases, corresponding well with the 46 cases reviewed by Weil (1931) who found that the site of lesion was cervical in 16 per cent, dorsal in 80 per cent, and lumbar in 4 per cent of cases.

The history of spinal symptoms before admission varied from 3 months to 2 years, the average period being 9 months, whilst the time that elapsed between the actual occurrence of signs of cord compression and admission to hospital varied from one week to one year.

Bony Involvement.—In 3 cases only did plain radiographs of the spine reveal any involvement of the vertebræ. In *Case* 2 there was porosis of a vertebral body with destruction of the pedicles and the presence of a right-sided soft-tissue shadow, in *Case* 6 there was definite erosion of a pedicle at the appropriate level, and in *Case* 10 there was scalloping of the posterior margin of the body of the involved vertebra.

TREATMENT

Nine cases were treated by decompressive laminectomy, the remaining case remaining untreated as the myelogram showed only an incomplete block, the diagnosis of Hodgkin's disease having been made by biopsy of an enlarged axillary lymph-node. All cases except two, one of which died before therapy, could be given and the other who had two courses of nitrogen mustard post-operatively, were given a course of deep X-ray therapy soon after the laminectomy wound had healed satisfactorily. The one case not subjected to laminectomy had two courses of nitrogen mustard.

Operation Findings.—Of the 9 patients treated by laminectomy, the tumour was found to be situated extradurally, was non-encapsulated, and was completely encircling the dural tube in 6 patients. In two the tumour was found mainly anteriorly and laterally, displacing the cord backwards and laterally, whilst in the last case there was no extradural intraspinal deposit, the cord being displaced backwards by expansion of the body of the vertebra from tumour tissue. In one case the tumour was found to have spread extraspinally and infiltrated the spinal musculature. In all cases with extradural tumour the neoplastic tissue could easily be stripped from the dura which did not itself appear to be infiltrated.

Results.—*Case* 8, who was admitted with very marked paraparesis, developed complete paraplegia 24 hours after operation and died 4 weeks later without any improvement. Case 5 who was admitted with complete paraplegia of 2 weeks' duration and died 6 weeks later without evidence of improvement. *Case* 6, who was admitted with complete paraplegia of 3 weeks' duration, also died 6 weeks later after having shown minimal improvement for the first 2–3 weeks. *Case* 3, who was admitted with complete paraplegia of 1 week's duration, died 3 weeks later with hardly any improvement. *Case* 4 showed an initial improvement with chemotherapy but died 3½ months later after deterioration in his general condition. *Case* 2 remained well for 1 year and 9 months, after which he is said to have died in a comatose condition, no autopsy being performed. *Case* 7 recovered power almost completely and remained well for 2 years and 1 month after which he died following a fit, no autopsy being performed. *Case* 9 improved remarkably and remains well a year and a half after operation. *Case* 10 has recovered considerable power and is well 3 months after operation. *Case* 1 is still leading a normal life 5 years and 4 months since operation.

The last 5 cases who showed considerable improvement with return almost to normality did not have a complete cord lesion at the time of treatment.

DISCUSSION

Elsberg (1942) found only one case of Hodgkin's disease in 253 spinal tumours, an incidence of 0·4 per cent. This series of 10 cases of Hodgkin's disease occurred in a total of 519 spinal tumours seen in a period of 23 years, an incidence of 1·9 per cent of all spinal tumours.

Though it is quite well known that involvement of the central nervous system in Hodgkin's disease is not uncommon, there are differing views as to the frequency of its occurrence. Smith and Stenstrom (1948), on reviewing the cases of Hodgkin's disease treated in the radiotherapy department of the University of Minnesota, found 11 cases with spinal involvement out of a total of 305 cases, an incidence of 3·6 per cent. Gray, Baker, Cottrell, and Skogland (1941) found 2 cases with cranial and 6 cases with spinal involvement out of a total of 79 cases of Hodgkin's disease, an incidence of 10 per cent. It may be quoted from Ginsburg that from 1914 to 1925 at the Montefiore Hospital in New York, 36 cases of Hodgkin's disease were under observation, in 3 of which spinal involvement was verified by biopsy or autopsy and in 2 of which such involvement was suspected clinically—an incidence of 14 per cent. Shapiro (1930) states that among 14 cases of Hodgkin's disease examined in the pathology laboratory at Cook County Hospital during 1929, 2 cases had shown disease of the spinal cord, again an incidence of 14 per cent.

The spinal symptoms in Hodgkin's disease can be produced in several ways. There may be an invasion of the vertebral body and its arch by lymphadenomatous tissue from the adjoining cervical, mediastinal, or retroperitoneal lymph-nodes which are close to the vertebral column. The symptoms may occasionally be produced by partial dislocation of the diseased vertebra causing mechanical spinal cord compression; McCallum (1931) reported a case of sudden death from dislocation of the diseased spine and severance of the cervical cord in Hodgkin's disease due to bony involvement. More commonly this invading lymphadenomatous tissue may infiltrate the vertebral body and then spread between the bone and dura anteriorly, subsequently surrounding the cord and causing cord compression as in *Case* 10. The neoplastic tissue may not, however, invade the extradural space but by causing expansion of a vertebral body can narrow the spinal canal and so give rise to cord compression as in *Case* 2.

More common, however, is the infiltration and invasion of the epidural space by lymphadenomatous tissue without any bony involvement. This spread occurs by extension from the adjacent lymph-nodes via the intervertebral foramina, the nerve-roots affording a channel; the extension occurring through the lymph-spaces of the nerve-roots rather than via the substance of the nerve-fibres. This type of extension is well seen in a case with cervical lymph-node enlargement where at first there was compression of the nerve-roots giving rise to root pains and weakness of the muscles supplied by these nerve-roots of the brachial plexus, and subsequently by the occurrence of paraparesis when lymphadenomatous tissue reached the spinal canal and caused cord compression. Weil (1931) in reviewing 46 cases of spinal cord lesions in Hodgkin's disease, found 4 per cent of cases showing syringomyelic changes, there being no evidence of cord compression by lymphadenomatous deposits. It can be easily understood that partial or total interruption of vascular supply to the spinal cord, if continued for a sufficient length of time, would produce a diffuse myelomalacia, the extent of these changes depending on the degree of interference with the blood-supply. If the interruption of the blood-supply be complete, it would lead to ischæmic softening of the involved segment of the cord. The addition of the stasis of lymphatic fluids at the segments involved would aggravate the interference with the normal nutrition of the nerve tissues. This interruption of the vascular supply may occur either within the intervertebral foramen or outside the spinal canal.

The 2 cases described by Allen and Mercer (1936) strongly support the view that cord changes may occur due to encephalomyelitis or myelitis and lymphocytic meningitis in Hodgkin's disease. In these cases, despite marked neurological deficit, no spinal, epidural or vertebral deposits or impairment of the

pulmonary metastases is described. Radio-active iodine tracer studies were of diagnostic value in the initial location of the carcinoma in the thyroid. The histology of the tumour and its metastases showed features in keeping with a carcinoma of relatively slow growth.

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40–49 yr.	2
50–59 yr.	4
60–69 yr.	2
70–79 yr.	2

Incidence of Primary Spinal Involvement.—Of these 10 cases only 2 had other manifestations of the disease and had been diagnosed as suffering from Hodgkin's disease before spinal symptoms appeared. The other 8 cases presented primarily with symptoms and signs of spinal involvement. Of these 8 cases only 2 were subsequently found to have developed other evidence of the disease, one having shown enlargement of lymph-glands 3 months later and the other having multiple deposits which were found at autopsy.

Site of Lesion.—The site of the spinal lesion was cervical in 1, dorsal in 7, and lumbar in 2 cases, corresponding well with the 46 cases reviewed by Weil (1931) who found that the site of lesion was cervical in 16 per cent, dorsal in 80 per cent, and lumbar in 4 per cent of cases.

The history of spinal symptoms before admission varied from 3 months to 2 years, the average period being 9 months, whilst the time that elapsed between the actual occurrence of signs of cord compression and admission to hospital varied from one week to one year.

Bony Involvement.—In 3 cases only did plain radiographs of the spine reveal any involvement of the vertebræ. In *Case* 2 there was porosis of a vertebral body with destruction of the pedicles and the presence of a right-sided soft-tissue shadow, in *Case* 6 there was definite erosion of a pedicle at the appropriate level, and in *Case* 10 there was scalloping of the posterior margin of the body of the involved vertebra.

TREATMENT

Nine cases were treated by decompressive laminectomy, the remaining case remaining untreated as the myelogram showed only an incomplete block, the diagnosis of Hodgkin's disease having been made by biopsy of an enlarged axillary lymph-node. All cases except two, one of which died before therapy, could be given and the other who had two courses of nitrogen mustard post-operatively, were given a course of deep X-ray therapy soon after the laminectomy wound had healed satisfactorily. The one case not subjected to laminectomy had two courses of nitrogen mustard.

Operation Findings.—Of the 9 patients treated by laminectomy, the tumour was found to be situated extradurally, was non-encapsulated, and was completely encircling the dural tube in 6 patients. In two the tumour was found mainly anteriorly and laterally, displacing the cord backwards and laterally, whilst in the last case there was no extradural intraspinal deposit, the cord being displaced backwards by expansion of the body of the vertebra from tumour tissue. In one case the tumour was found to have spread extraspinally and infiltrated the spinal musculature. In all cases with extradural tumour the neoplastic tissue could easily be stripped from the dura which did not itself appear to be infiltrated.

Results.—*Case* 8, who was admitted with very marked paraparesis, developed complete paraplegia 24 hours after operation and died 4 weeks later without any improvement. Case 5 who was admitted with complete paraplegia of 2 weeks' duration and died 6 weeks later without evidence of improvement. *Case* 6, who was admitted with complete paraplegia of 3 weeks' duration, also died 6 weeks later after having shown minimal improvement for the first 2–3 weeks. *Case* 3, who was admitted with complete paraplegia of 1 week's duration, died 3 weeks later with hardly any improvement. *Case* 4 showed an initial improvement with chemotherapy but died 3½ months later after deterioration in his general condition. *Case* 2 remained well for 1 year and 9 months, after which he is said to have died in a comatose condition, no autopsy being performed. *Case* 7 recovered power almost completely and remained well for 2 years and 1 month after which he died following a fit, no autopsy being performed. *Case* 9 improved remarkably and remains well a year and a half after operation. *Case* 10 has recovered considerable power and is well 3 months after operation. *Case* 1 is still leading a normal life 5 years and 4 months since operation.

The last 5 cases who showed considerable improvement with return almost to normality did not have a complete cord lesion at the time of treatment.

DISCUSSION

Elsberg (1942) found only one case of Hodgkin's disease in 253 spinal tumours, an incidence of 0·4 per cent. This series of 10 cases of Hodgkin's disease occurred in a total of 519 spinal tumours seen in a period of 23 years, an incidence of 1·9 per cent of all spinal tumours.

Though it is quite well known that involvement of the central nervous system in Hodgkin's disease is not uncommon, there are differing views as to the frequency of its occurrence. Smith and Stenstrom (1948), on reviewing the cases of Hodgkin's disease treated in the radiotherapy department of the University of Minnesota, found 11 cases with spinal involvement out of a total of 305 cases, an incidence of 3·6 per cent. Gray, Baker, Cottrell, and Skogland (1941) found 2 cases with cranial and 6 cases with spinal involvement out of a total of 79 cases of Hodgkin's disease, an incidence of 10 per cent. It may be quoted from Ginsburg that from 1914 to 1925 at the Montefiore Hospital in New York, 36 cases of Hodgkin's disease were under observation, in 3 of which spinal involvement was verified by biopsy or autopsy and in 2 of which such involvement was suspected clinically—an incidence of 14 per cent. Shapiro (1930) states that among 14 cases of Hodgkin's disease examined in the pathology laboratory at Cook County Hospital during 1929, 2 cases had shown disease of the spinal cord, again an incidence of 14 per cent.

The spinal symptoms in Hodgkin's disease can be produced in several ways. There may be an invasion of the vertebral body and its arch by lymphadenomatous tissue from the adjoining cervical, mediastinal, or retroperitoneal lymph-nodes which are close to the vertebral column. The symptoms may occasionally be produced by partial dislocation of the diseased vertebra causing mechanical spinal cord compression; McCallum (1931) reported a case of sudden death from dislocation of the diseased spine and severance of the cervical cord in Hodgkin's disease due to bony involvement. More commonly this invading lymphadenomatous tissue may infiltrate the vertebral body and then spread between the bone and dura anteriorly, subsequently surrounding the cord and causing cord compression as in *Case* 10. The neoplastic tissue may not, however, invade the extradural space but by causing expansion of a vertebral body can narrow the spinal canal and so give rise to cord compression as in *Case* 2.

More common, however, is the infiltration and invasion of the epidural space by lymphadenomatous tissue without any bony involvement. This spread occurs by extension from the adjacent lymph-nodes via the intervertebral foramina, the nerve-roots affording a channel; the extension occurring through the lymph-spaces of the nerve-roots rather than via the substance of the nerve-fibres. This type of extension is well seen in a case with cervical lymph-node enlargement where at first there was compression of the nerve-roots giving rise to root pains and weakness of the muscles supplied by these nerve-roots of the brachial plexus, and subsequently by the occurrence of paraparesis when lymphadenomatous tissue reached the spinal canal and caused cord compression. Weil (1931) in reviewing 46 cases of spinal cord lesions in Hodgkin's disease, found 4 per cent of cases showing syringomyelic changes, there being no evidence of cord compression by lymphadenomatous deposits. It can be easily understood that partial or total interruption of vascular supply to the spinal cord, if continued for a sufficient length of time, would produce a diffuse myelomalacia, the extent of these changes depending on the degree of interference with the blood-supply. If the interruption of the blood-supply be complete, it would lead to ischæmic softening of the involved segment of the cord. The addition of the stasis of lymphatic fluids at the segments involved would aggravate the interference with the normal nutrition of the nerve tissues. This interruption of the vascular supply may occur either within the intervertebral foramen or outside the spinal canal.

The 2 cases described by Allen and Mercer (1936) strongly support the view that cord changes may occur due to encephalomyelitis or myelitis and lymphocytic meningitis in Hodgkin's disease. In these cases, despite marked neurological deficit, no spinal, epidural or vertebral deposits or impairment of the

Table II.—SUMMARY OF 10 CASES OF SPINAL COMPRESSION IN HODGKIN'S DISEASE

SEX AND AGE	SYMPTOMS AND SIGNS	OTHER MANIFESTATIONS OF DISEASE	RADIOGRAPHY OF SPINE	OPERATIVE FINDINGS	TREATMENT	FOLLOW-UP
1. E. S. S. F. 47	4 mth. girdle pain 6 wk. paraparesis 1 wk. urgency of micturition On Examination: Marked spastic paraparesis, right worse than left, with sensory impairment up to D4	Nil	N.A.D. Myelogram: Extradural tumour causing complete block at D4/5 level	Extradural firm grey tumour completely encircling the cord and intimately related to the nerve-roots	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	Completely well 5 yr. 4 mth. later
2. E.B. M. 59	8 yr. previously backache and right sciatica Biopsy of neck lymph-node and deep X-ray therapy then and 2 yr. later with marked relief 1 yr. of recurrence of pain and difficulty in walking On Examination: Wasting of lower limbs with weakness of dorsiflexion and eversion of both feet	Diagnosed as Hodgkin's disease 8 yr. previously Nil now	Porosis and flattening of body of L4 vertebra, erosion of pedicles and spinous process and soft-tissue mass on the right side at same level	Infiltration of the lamina, pedicles, and spinous process; cord pushed back by tumour in the body but no extra-dural intraspinal tumour	*a.* Decompressive laminectomy *b.* Two courses of nitrogen mustard, then and a year later	Relieved of his pain, left only with slight residual weakness and able to do a light job for 1 yr. 9 mth. when died in coma No autopsy performed
3. G. F. M. 41	1 yr. 6 mth. previously backache, pyrexia of unknown origin, diagnosed as Hodgkin's disease and treated with deep X-ray therapy 6 mth. progressive weakness of legs 3 wk. ago improvement with deep X-ray therapy. 1 wk. rapid complete paraplegia with retention On Examination: Complete spastic paraplegia with sensory level at D7	Diagnosed as Hodgkin's disease 1 yr. 6 mth. previously. Few nodes palpable now in the axilla	N.A.D. Myelogram: Extradural tumour with complete block at D6/7 from posteriorly	Firm pale extradural tumour covering the cord posteriorly and laterally	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	Had only slight recovery of sensation initially Died 3 wk. later. No autopsy performed
4. G. B. M. 55	1 yr. backache between shoulder-blades 9 mth. progressive weakness of legs On Examination: Spastic para-paresis, left more than right with probable sensory impairment up to mid-dorsal region	Axillary and cervical nodes palpable on subsequent admission and pyrexia	N.A.D. Myelogram: Incomplete block at D6 level	Biopsy of axillary lymph-node —Hodgkin's disease	*a.* Two courses of nitrogen mustard	Improved with initial course of chemotherapy but relapsed soon and died 5 mth. later P.M. Findings: Manifestation in various lymph-nodes and persistence of cord compression at L3 level by Hodgkin's deposit
5. E. A. C. F. 75	1 yr. of progressive weakness of legs 3 mth. weakness more marked with occasional flexor spasm 1–2 wk. complete paraplegia with sphincter disturbances On Examination: Complete spastic paraplegia with sensory level up to D4/5	Nil	N.A.D. Myelogram: Extradural tumour at D3 level with complete block	Firm extradural tumour completely surrounding the dural tube—easily stripped off the dura	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	No improvement Died 6 wk. later. No autopsy performed
6. J. C. M. 76	5 mth. backache and pain both legs 3 mth. weakness of legs 3 wk. paraplegia 1 wk. retention of urine On Examination: Complete para-plegia with sensory level up to L3	Nil	Erosion of right pedicle of D11 vertebra Myelogram: Extradural tumour at D11–12 level with complete block	Firm, grey, extradural tumour, situated mainly posteriorly and laterally	*a.* Decompressive laminectomy	Slight recovery of power initially Died 6 wk. later P.M. Findings: Foci in spleen; affected nodes in neck, porta hepatis; upper surface of pancreas, etc.
7. M. K. F. 60	3 mth. neck ache with pain radiating down the shoulders 1 mth. weakness of hands and legs and sphincter disturbances On Examination: Weakness of muscles of hand, elbow and both legs, sluggish biceps jerk, brisk other reflexes, sensory impairment up to nipples	Nil	N.A.D. except for spondylosis at C5/6 level Myelogram: Extradural tumour at C6 level with complete block	Firm, extradural tumour situated laterally and mainly anteriorly pushing the cord backwards	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	Returned to normality except for minimal weakness of left hand Died 2 yr. 1 mth. later after having had a fit. No autopsy performed

Table II.—Summary of 10 Cases of Spinal Compression in Hodgkin's Disease—*continued*

Sex and Age	Symptoms and Signs	Other Manifestations of Disease	Radiography of Spine	Operative Findings	Treatment	Follow-up
8. E. L. M. 61	7 mth. girdle-like pain D7–9 segments for 3 wk. 3 wk. recurrence of pain and progressive weakness of legs On Examination: Marked spastic paraparesis, right worse than left, with sensory impairment up to D8 level	Nil	N.A.D. Myelogram: Extradural tumour at D7 level with complete block, mainly from left side	Firm, extradural tumour enveloping the cord completely	*a.* Decompressive laminectomy *b.* Deep X-ray therapy for 2 wk.	Developed complete paraplegia 24 hr. after operation. Died 4 wk. later. No evidence of tumour; infected bed-sores and pyelonephritis
9. A. K. M. 55	6 mth. pain mid-dorsal region and right costal margin 3 mth. numbness below waist and progressive paraparesis On Examination: Moderate spastic paraparesis, left worse than right with sensory impairment up to D10 level	Nil	N.A.D. Myelogram: Extradural tumour at D10/11 displacing the cord anteriorly and to the right with complete block	Firm, extradural tumour, completely surrounding the dural tube	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	Quite well 1 yr. 6 mth. later, working normally
10. J. F. F. 56	2 yr. of low backache and left-sided sciatic pain 2–3 wk. marked weakness and œdema of left leg On Examination: Marked œdema of left leg; marked generalized weakness of left leg with foot-drop and some weakness of right foot	Nil	Erosion of posterior margin of body of L3 vertebra Myelogram: Extradural lesion pushing the dural tube to the right side at L3 level with complete block	Firm, extradural tumour extending extraspinally and infiltrating the spinal musculature; lying mainly anteriorly and on the left side	*a.* Decompressive laminectomy *b.* Deep X-ray therapy	Markedly improvement immediately and 3 mth. later

vascular supply were detected. There was an extensive œdema of the cord, especially at the periphery and around blood-vessels, engorgement of blood-vessels, infiltration of the nervous tissue with small round cells, and changes in the nerve-cells. Cerebrospinal fluid was xanthochromic with increased protein, excess of globulin, and some small round cells. The appearances were highly suggestive of lymphocytic meningitis with perivascular cuff-like lymphocytic infiltration.

Winkelman and Moore (1941) report a case of Hodgkin's disease with both spinal and cranial involvement in whom the dura as well as both cerebral hemispheres were affected, the intracerebral deposit being completely separate and distinct from the dural deposit. They therefore state that it was almost certainly a hæmatogenous spread of lymphadenomatous tissue into the cerebral hemisphere unless one postulated the extension of the tissue from the dura along pial vessels. Martin and Courville (1936) also believe that hæmatogenous deposit of lymphadenomatous tissue can occur in the spinal epidural space.

Kubre in 1927 showed that nests of lymphocytes did exist in the spinal epidural space. Verda (1944) agrees with Kubre that these nests of lymphocytes could be the primary site of affection in Hodgkin's disease, thus leading to the primary formation of lymphadenomatous tissue in the spinal epidural space and not by extension from the adjacent lymph-nodes. This certainly can account for the production of neurological disturbances alone without any other manifestation of the disease. This conception is certainly exemplified by *Cases* 1, 7, 8, 9, 10 of this series in whom the only sign of the disease had been spinal, *Case* 1 being alive and well 5 years and 4 months after treatment and still not showing any spread of the disease. It is important to recognize this entity—as it is in these cases that one may hope to obtain a prolonged period of symptom-free existence, the treatment being directed, as it were, to a localized rather than a generalized disease.

The substance of the cord rarely becomes invaded by lymphadenomatous tissue; the dura offering a firm resistance but invasion of the subdural space and cord elements may occur occasionally along the septa of the nerve-root.

The management of a case would depend on how it presents itself. Either it would present as a case of known Hodgkin's disease with spinal manifestations, or as one of spinal cord compression of unknown aetiology. When it is reasonably certain that lymphadenoma is the cause of the spinal symptoms, and if there is a complete manometric and myelographic block, a decompressive laminectomy followed by local deep X-ray therapy or possibly chemotherapy would seem to be the treatment of choice. If, however, the spinal cord compression be incomplete and there is no doubt of the aetiology, a case might be made out for local deep X-ray therapy or chemotherapy without preliminary decompressive laminectomy; for *Case* 4 in this series was treated with some temporary relief in a similar way. When the exact nature of the lesion causing spinal cord compression is not definitely known, a laminectomy should always be performed to establish the correct diagnosis. There were 6 cases in our series over the age of 50

without any generalized manifestations of Hodgkin's disease, 2 showing erosion of the bony canal in plain X-rays and all having a myelographic appearance of extradural tumour which could easily have been mistaken clinically for secondary carcinomatous deposits but for laminectomy and histopathological examination of the neoplastic tissue.

The object of the treatment is obviously to prevent the occurrence of paraparesis or paraplegia, or to relieve it if it has already occurred. When complete paraplegia is produced, the longer the cord compression is allowed to persist the smaller the chance of recovery, which emphasizes the need for prompt treatment. If the treatment is given early enough, there is no doubt that a reasonable recovery may be hoped for, leading to several months or years of comfortable existence as seen in *Cases* 1, 2, 4, 7, 9, and 10. Lawes and Ham (1953) report a typical case of Hodgkin's disease with quadriplegia of 48 hours' duration who responded very satisfactorily to chemotherapy and was alive and well 6 months later.

Weil (1931) found that, once the neurological symptoms appeared, over 80 per cent of his cases died within 3 months of their appearance and none survived more than one year after the onset of paralysis. Sparling, Adams, and Parker (1947) also felt that the course of Hodgkin's disease is shortened considerably by the advent of spinal paraplegia, though isolated cases were observed to have survived for 1–2 years. However, Jackson and Parker (1947) found that 3 of their 10 patients with paraplegia survived for over one year, 1 being alive and in good health 4 years later. Of our 2 cases who were diagnosed and treated primarily as Hodgkin's disease and who developed spinal symptoms at a later date, one (*Case* 3) died within 9 months of the occurrence of spinal symptoms, whereas the other (*Case* 2) lived for nearly 9 years after the occurrence of spinal symptoms. Of 2 other cases who were subsequently found to have had other manifestations of the disease, one (*Case* 6) died within 4½ months and the other (*Case* 4) about 16 months after the onset of symptoms of cord compression.

From the analysis of these cases there seems to be little doubt about the truth in Ginsburg's statement that in Hodgkin's disease "the nervous system may become clinically, primarily and predominantly affected and give rise to symptoms for months and years before the occurrence of any demonstrable enlargement of the lymph-nodes and spleen". Believing in the existence of nests of lymphocytes in the spinal epidural space, we feel inclined to go a step further and state that, if diagnosed and treated adequately and sufficiently early, there might not be a further spread of this spinal epidural lymphadenomatous deposit. It is also quite clearly shown that the prognosis is certainly better when the spinal canal is the region of "primary involvement" and not due to secondary extension from adjoining lymph-nodes.

SUMMARY

1. Ten cases of Hodgkin's disease with spinal deposits are analysed, the cases forming a part of 519 spinal tumours admitted under one of us (W. McK.) from 1937 to 1960, thus representing an incidence of 1·9 per cent of all spinal tumours.

2. The various modes of involvement of the spinal canal by lymphadenomatous tissue and the mechanisms of production of spinal symptoms are discussed.

It is stressed that the nervous system may be primarily and predominantly affected in Hodgkin's disease and give rise to symptoms many months or years before any other manifestations of the disease may appear.

3. Need to obtain a correct diagnosis, by laminectomy if necessary, in cases of spinal cord compression is stressed to enable one to offer appropriate treatment.

4. Decompressive laminectomy in cases with complete manometric and myelographic block followed by deep X-ray therapy locally is recommended, chemotherapy being offered for generalized manifestations. With this therapy, reasonable survival for months or years was achieved in 6 out of the 10 patients in this series. The longest survival in this series was 5 years and 4 months, the patient being still alive and well.

5. The prognosis is found to be definitely better in cases with primary spinal involvement and no spread of the disease than in cases with spinal involvement due to secondary extension from adjoining lymph-nodes.

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EXPERIMENTAL SURGERY

REPLACEMENT OF VENOUS DEFECTS

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BECAUSE of the rich alternate venous pathways in most parts of the body, indications for replacement of veins will always be fewer than those for replacement of arteries. However, lesions of vital veins, such as the superior and inferior venæ cavæ, the portal vein, and the renal vein, continue to arouse interest in their repair. Furthermore, the development of a successful experimental technique of venous replacement might eventually lead to replacement of damaged extremity veins containing functioning valves (DeWeese and Niguidula, 1960). Clinical experience of the replacement of venous defects reported thus far has been confined to attempts to replace or bypass the superior vena cava in cases of the superior vena cava syndrome (Ashburn, Sewell, and Huggins, 1956; Allansmith and Richards, 1958; Deterling and Bhonslay, 1955; Hanlon, 1954; Holman and Sternberg, 1954; Kay, 1954; Klassen, Andrews, and Curtis, 1951; Samson, 1954; Scannell and Shaw, 1954), one reported case of inferior vena cava replacement (Blum, Medl, and Keefer, 1956), and another of substitution of the portal vein (Kikuchi, 1956). Grafts have been used for splenorenal anastomoses (Rousselot, 1952) and for portocaval shunts (Walker, 1954).

EXPERIENCE WITH GRAFTS IN THE VENOUS SYSTEM IN EXPERIMENTAL ANIMALS

Aortic homografts have been used to replace the superior vena cava generally with poor results (Ashburn and others, 1956; Allansmith and Richards, 1958; Bryant, Lazerty, and Howard, 1958; Deterling and Bhonslay, 1955; MacLean, Phibbs, Flom, and Brainard, 1949; Ohara and Sakai, 1957; Schauble, Anylan, and Postlewait, 1959). Aortic homografts are technically easy to use, but a high proportion thrombose early, and of those that remain patent in the early stages many become furrowed and contracted before eventual closure (Deterling and Bhonslay, 1955). Attempts at replacement of the inferior vena cava by arterial homograft have met with little success (Bryant and others, 1958; Ohara and Sakai, 1957). Bryant found all 8 of his trial grafts closed after a short interval. Creation of experimental shunts between the portal vein and the vena cava have also met with unsatisfactory results (Southwick, 1950). Autogenous aorta, however, appears to be an excellent venous substitute (Moore and Riberi, 1958; Moore, 1960), Moore having found all of 31 canine superior vena cava replacements open at a later study.

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Venous Autografts.—Replacement of the jugular and femoral veins has been reported to carry a patency rate varying from 50 per cent to 85 per cent (DeWeese and Niguidula, 1960; Earle, Horsley, Villavicencio, and Warren, 1960; Schauble, 1959) and autogenous vein appears to be the best substitute for the cavæ in the dog (Sauvage and Gross, 1960). Defects in the portal circulation have been bridged with autogenous vein (Daniel, 1952), but the results were poor, only 4 grafts out of 13 remaining patent for 2 months. In infant pigs, however, good results have been reported (Sauvage and Wesolowski, 1955) as the autogenous vein replacement for the cava grew with the animals.

Plastic Prostheses.—A large variety of plastic materials has been used in the experimental replacement of the veins, amylon (similar to nylon) and silicone rubber (Ohara and Sakai, 1957), polyvinyl sponge (Benjamin, Turbak, Hannon, Shumway, and Lewis, 1957), polyethylene (Egdahl and Hume, 1956), nylon (Deterling and Bhonslay, 1955), orlon (Bryant and others, 1958), dacron (Deterling and Bhonslay, 1955), teflon (Bryant and others, 1958), lucite (Daniel, 1952), and teflon sponge (Botham, Dracopoulas, and Gale, 1960). Only the last named has given a high success rate—12 of 16 grafts were patent between 4 and 26 weeks later.

Other Substitutes.—Pedicle tubes fashioned from the right atrium have been successfully used to replace small sections of the canine superior vena cava (Riberi and Moore, 1959), but the method is of limited application.

It was clear from these studies that there were two major factors to be considered in choosing a graft for placement in the venous system—namely, tissue reaction to the graft and the rigidity of the graft walls. Autogenous veins are the best substitute as far as tissue reaction is concerned, but homologous arteries and fabric prostheses are more resistant to collapse. Occasionally, such as in the venæ cavæ, an autogenous vein of adequate size is not readily available but isolated experiences with the compilation technique has shown its promise as a means of overcoming this difficulty (Deterling and Bhonslay, 1955; Earle and others, 1960). It was therefore used in the present study, which was designed to examine further the technical factors involved in successful replacement of the vena cava.

The abdominal inferior cava of the dog was chosen for replacement, because after anastomosis of the renal vein to the graft post-operative renal function provided a good indicator of graft patency. Also the need for renal vein anastomosis allowed us to test the feasibility of anastomosing tributary veins to the side of the graft. The influence of kinking and external pressure on the grafted segment has also been tested.

MATERIAL AND METHODS

Healthy mongrel dogs of weights varying between 12 and 25 kilograms were used and anæsthetized with pentobarbital sodium (nembutal) in doses of approximately 30 mg./kilo administered by the intravenous route.

The first 11 dogs were females, and a preliminary episiotomy was done so that the conscious dog could be later catheterized for urinary studies. The remaining dogs were of both sexes. Urinary studies were not attempted on these because the first eleven showed that serial estimations of the blood-urea nitrogen gave an accurate index of graft patency.

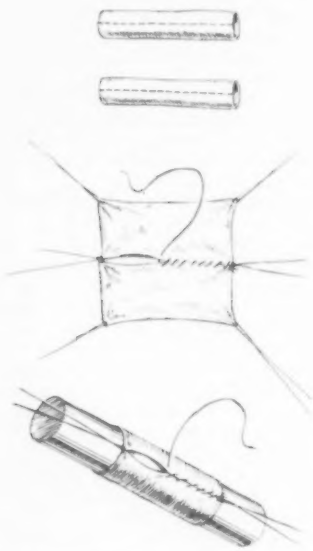


FIG. 790.—Diagram showing method of fashioning compilation grafts.

At the beginning of each operation an intravenous saline drip was set up in a convenient leg vein. Through vertical incisions in the neck approximately 5–6 cm. were excised from both external jugular veins after careful stripping of the adventitia. Small tributaries were not ligated at the jugular end, as ligatures interfered with subsequent operative manœuvres, and bleeding from these veins was never a problem. Forceps were not applied to the veins at any stage and they were retracted only by moistened tape. Robertson (1960) has recently stressed the effect of minor trauma to the intima as a cause of thrombosis in the venous system.

Both veins were laid side-to-side on moist gauze, split longitudinally with fine scissors, and inspected for the presence of valves, which are often present in the canine external jugulars. If valves were present, the split veins were laid so that they did not interfere with the direction of flow in the graft. Holding sutures of 5–0 arterial silk were placed at each corner and the veins were slung by them from a simple rectangular metal frame as described by Earle and others (1960). The veins were suspended so that they lay side by side, with their intimal surface facing downwards. The adjacent sides were then sutured with 5–0 silk in a continuous over-and-over suture, bites being taken at intervals of about 1 mm.

The graft was then turned so that the endothelial surface faced upwards, and a glass tube of approximately the same diameter as the width of one segment of the graft was laid longitudinally on the intimal surface of one component and the other vein folded over to form a tube. The glass tube acted as a prosthesis to avoid variation in the graft diameter while the remaining edge was sutured (*Fig.* 790).

The abdomen was opened through a long midline incision and the inferior vena cava exposed and cleared of peritoneum from just below the liver to the bifurcation.

Right nephrectomy was performed and the left adrenal vein, the right gonadal vein, and the lumbar veins divided between ligatures.

At this stage of the operation the intravenous saline infusion was replaced by one containing 20 mg. heparin to 500 ml. saline, and this was infused at about 20 drops a minute for the remainder of the procedure.

The left renal vein was defined and the inferior vena cava divided well above the renal vein between proximal and distal sheathed bulldog clamps. The graft was arranged so that the seams lay directly anterior and posterior, and two mattress sutures of 5–0 silk were inserted between the graft and the upper cava in these positions so that the graft lay without kinking. The anastomosis was completed by a continuous over-and-over suture on each side.

An oval aperture was cut in the left side of the graft in the same position and at the same angle as the renal vein met the cava. A bulldog clamp was placed on the renal vein and another on the cava below the site of the renal vein graft anastomosis. The renal vein was divided at its junction with the cava. The renal vein to graft anastomosis was made with 5–0 silk, a mattress suture at the superior and inferior angles, and a continuous over-and-over suture between them. The clamps on the inferior vena cava above and the renal vein were removed to restore renal vein flow. The lower cava was then excised and the lower anastomosis completed (*Fig.* 791). Finally the graft was covered with peritoneum and the abdomen closed.

A similar procedure was carried out using a standard crimped woven teflon prosthesis as a replacement to provide a comparison between autogenous vein and fabric prostheses as a replacement for the inferior vena cava. Because of difficulty in obtaining good apposition between the cava and the graft with a continuous over-and-over suture the anastomoses in these grafts were done with continuous mattress sutures of 5–0 silk.

Prior to the use of a heparin drip during the procedure thrombosis commonly occurred in the occluded lower cava and had to be flushed out before completion of the lower anastomosis. After the routine use of heparin thrombosis in this segment was not seen.

Post-operatively the dogs were warmed by blankets. Failure to do this in the preliminary work

resulted in the death of 5 dogs among the 11 animals in the series which died at operation or were found dead in their cages the next day.

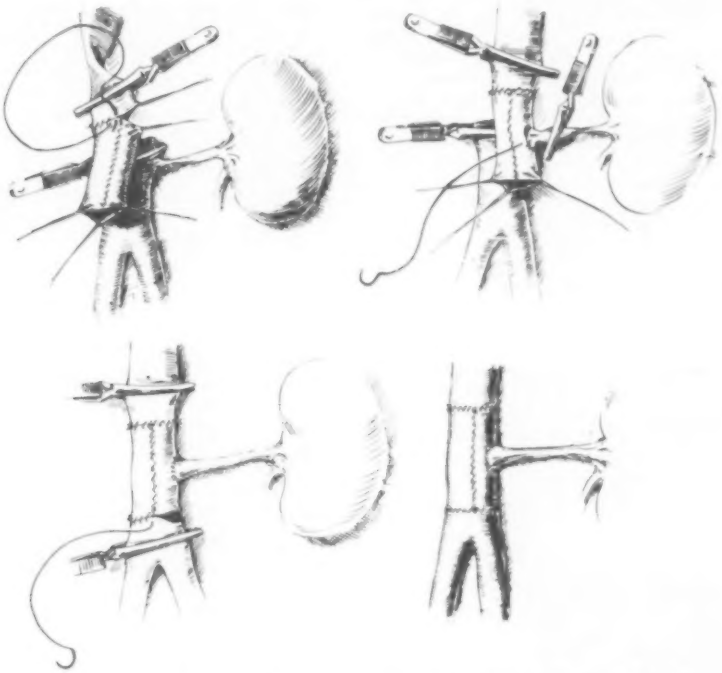


FIG. 791.—Stages in replacement of the inferior vena cava with compilated autogenous vein graft.

The pressures in the vena cava below the first clamp were determined in 4 dogs in an attempt to decide whether a useful amount of renal vein blood returns to the heart via collaterals from the lower cava during the period of occlusion. Pressure measurements confirmed the findings of Schauble and others (1959) that the pressure in the occluded cava rapidly built up to between 70 cm. and 100 cm. of water, but then began to fall so that 20 minutes later it is between 40 cm. and 50 cm. of water (*Fig.* 792). These findings suggested that sufficient venous collaterals function in the time required for operation to allow useful drainage from the kidney after occlusion of the inferior vena cava.

A total of 42 grafts were used. In 4 dogs the inferior vena cava was replaced from below the renal veins to the bifurcation. In 26 dogs the subhepatic inferior vena cava was replaced with compilated autogenous jugular vein grafts with anastomosis of the left renal vein to the graft, and in 12 dogs crimped woven teflon prostheses replaced a similar portion of the cava. Right nephrectomy was done in all 38 dogs. The animals were followed for varying periods of time up to six months after operation.

The state of patency of the grafts in the post-operative period was determined by observation of the function of the remaining kidney. Initially this was attempted by phlebography, but phlebograms showed the caval replacement only and gave no information about the renal vein. Studies were carried out on the albumin and cellular content of the urine and the blood-urea nitrogen in 11 dogs. Serial blood-urea nitrogen estimations proved a satisfactory guide to graft patency, and this was the sole post-operative test used in the remaining experiments.

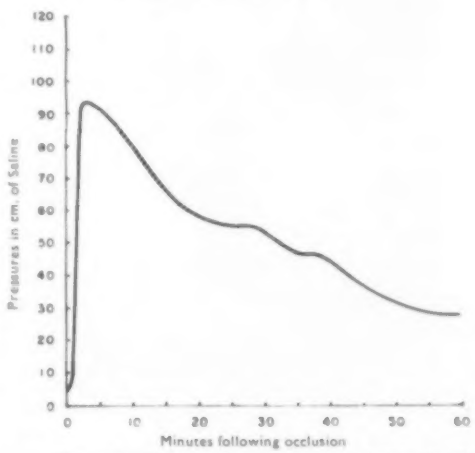


FIG. 792.—Diagram showing pressure changes in the external iliac vein of the dog following occlusions of the inferior vena cava.

RESULTS

Effects of Closure of the Graft.—Dogs with patent grafts showed a characteristic pattern of blood-urea nitrogen estimations in the post-operative period. The level which varied between 9 mg. /100 ml. and 18 mg. /100 ml. before operation rose to 35–45 mg. /100 ml. on the 2nd post-operative day and rapidly fell to normal by the 7th to the 14th day. In dogs with closed grafts, however, there was a rapid rise of blood-urea nitrogen to 50–60 mg. /100 ml. on the 1st post-operative day and the rise continued until a level of 100–130 mg. /100 ml. was reached by the 8th to 12th day. In dogs which survived it fell towards normal levels on the 18th to 20th day (*Fig.* 793). Two dogs with thrombosed grafts failed to survive more than 4 days, and at autopsy the appearance of the kidney was identical with that observed after ligation of the renal vein. It was three times its normal size, deep blue in colour, with large subcapsular hæmatomas, capsular tears, and perirenal extravasations of blood. In 1 of these dogs thrombus had extended from the graft into the renal and iliac veins and a mechanical obstruction of the renal vein itself was present in the second animal.

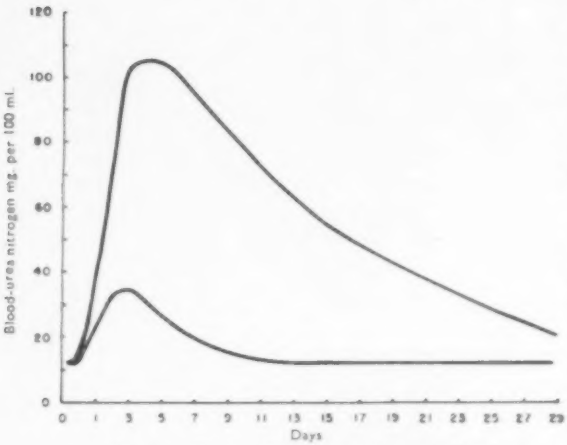
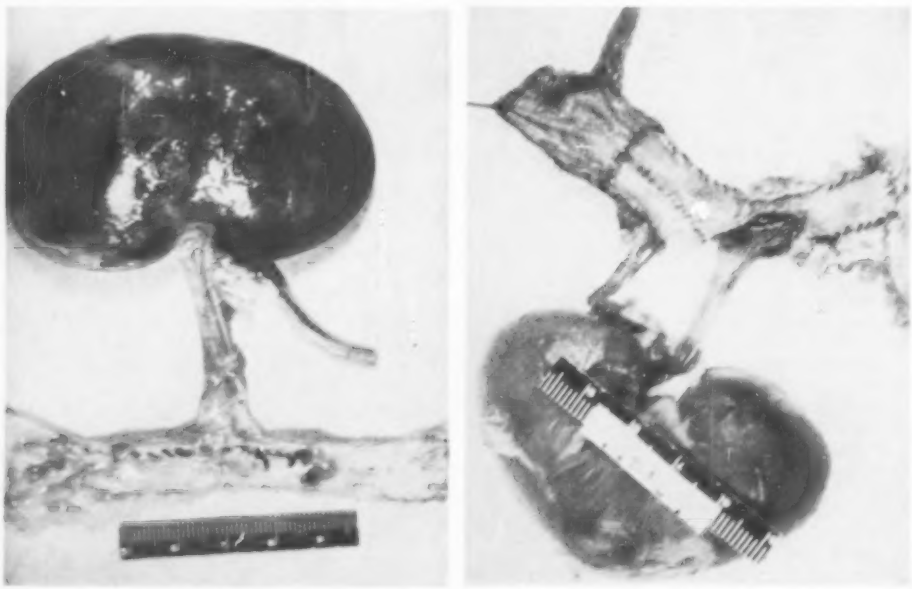


FIG. 793.—Diagram showing mean changes in blood-urea nitrogen in 18 dogs after replacement of the inferior vena cava (lower curve mean of 13 open grafts, upper curve mean of 5 closed grafts).

All the dogs were autopsied. Patent vein grafts were indistinguishable from normal vena cava except for the suture lines (*Fig.* 794) and the kidneys were macroscopically and microscopically normal. Dogs with closed grafts which survived more than a week showed abundant venous collaterals in the perirenal tissues and the abdominal wall.

All the patent teflon grafts were lined with compressed fibrin. There was evidence of ingrowth of living intima only immediately adjacent to the anastomoses. In the long-term survivors this layer was very thin, and the replacements were surrounded by an intense fibrous reaction. This outer layer stripped easily at autopsy, and the underlying graft appeared unchanged. The kidneys in these three dogs were also histologically normal.



A B

FIG. 794.—A, Autogenous vein graft in inferior vena cava of the dog at autopsy 118 days after operation. B, Inferior vena cava of dog replaced with autogenous vein compilation graft 107 days after operation. Cava and graft opened longitudinally to show anastomoses.

Early Deaths.—Eleven dogs either died at operation or were found dead in the cage the following morning, 7 in the compilated vein group and 4 in the

teflon group; they are not included in the detailed analysis of the study. Deaths were due to technical failure in 3 dogs, excessive hæmorrhage in 1, respiratory failure in 2, and hypothermia in 5. Studies during the course of two later experiments showed that the temperature of the dogs fell to 82–85° F. by the end of the operation, owing partly to the effect of nembutal and partly to the low temperature at which the air conditioning apparatus had been set. All subsequent dogs were routinely warmed post-operatively, and there were no further deaths from this cause. There were 4 patent grafts among the 7 dogs found dead the morning after operation.

FIG. 795.—Inferior vena cava of dog sacrificed 193 days after replacement of a segment with autogenous vein compilation graft showing upper anastomosis covered with endothelium.

Survivors.—The results of replacement of the inferior vena cava in all 42 dogs are analysed in *Table I*. The results in the surviving dogs with replacement of the inferior vena cava and renal vein anastomosis are analysed in detail in *Table II*. In the absence of comment to the contrary the term 'patent' describes a graft without thrombosis, fibrosis, or narrowing at the suture lines in an animal in which the renal vein and kidney were normal.

Replacement of the Post-renal Inferior Vena Cava with Compilated Autogenous Vein Graft.—Four dogs were operated upon initially without renal vein anastomosis before much experience had been obtained in the technique of vena caval replacement, and all the grafts thrombosed shortly after operation.

Replacement of the Subhepatic Inferior Vena Cava by Compilated Autogenous Vein Grafts and Anastomosis of the Renal Vein to the Graft.—Twelve dogs had patent grafts which resembled the normal vena cava; 1 graft was patent with a normal renal vein but was fibrosed and constricted by fibrous tissue at the anastomosis. The 6 closed grafts which thrombosed did so in the immediate post-operative period. Three of them had well-formed collaterals; the others died before collaterals had time to form. The kidney in those dogs that had no collaterals was pale and œdematous and about twice as large as normal; the dogs with good collaterals had normal kidneys.

Replacement of Subhepatic Inferior Vena Cava with Crimped Woven Teflon and Anastomosis of the Renal Vein to the Graft.—In 4 successful dogs the grafts were covered with tough fibrous tissue and the renal vein and kidney were histologically normal. The fibrous tissue was adherent to surrounding structures but stripped easily from the graft. Two grafts which closed immediately after operation had findings similar to those seen after renal vein ligation; the other 2 showed fibrosis and contracture at the anastomoses with normal kidneys.

DISCUSSION

The ideal replacement for the veins should carry a good immediate and long-term patency rate, at least as good as that for grafts used in arterial surgery. It should take sutures well, be convenient to handle, and should be easily available and easily prepared for use. Grafts placed in the venous system have a lower immediate and long-term success rate than have similar grafts used to replace the arteries. As Bryant and his co-workers (1958) showed, this is so because of the low pressure in the system and the consequent slow rate of flow which encouraged thrombosis initiated by minor constrictions and roughenings which would be of little consequence in the rapidly flowing arterial system. Careful technique is therefore more essential in operations on veins than on arteries. Veins are difficult to handle, because their walls are thin, friable, and non-rigid, and the cut ends of the vein tend to retract and collapse. The intima and media of the vein are but loosely attached to one another so that one or more layers may be missed when

Table I.—RESULTS OF EXPERIMENTAL REPLACEMENT OF THE INFERIOR VENA CAVA OF THE DOG

TYPE OF GRAFT	AUTOGENOUS VEIN COMPILATION GRAFTS		CRIMPED WOVEN TEFLON	TOTALS
Anatomical Replacement	Subrenal IVC	Subhepatic IVC with Renal Vein Anastomosis	Subhepatic IVC with Renal Vein Anastomosis	
Number of dogs	4	26	12	42
Number of post-operative deaths	0	7	4	11
Number patent	0	13	4	17
Number closed	4	6	4	14
Percentage patent in survivors	0	70	50	55

RESULTS

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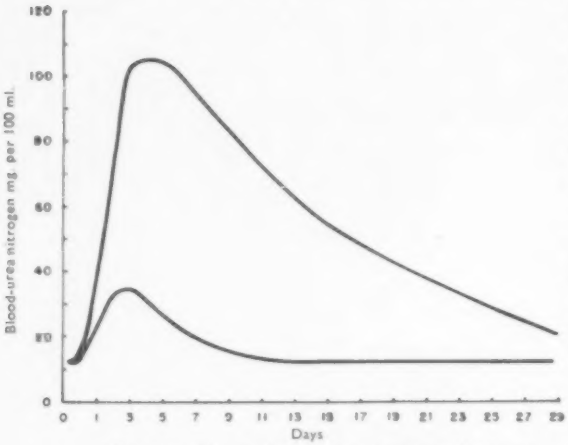

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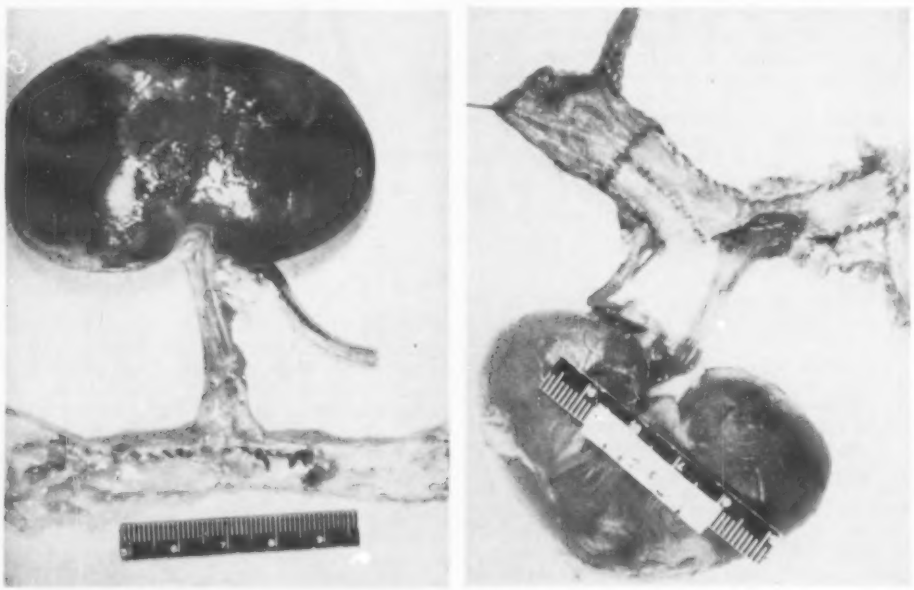

A B

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sutures are placed. The size of a graft in relation to the parent vein is also important, because any disproportion in length or diameter leads to angulation, buckling, and ridging, and this further disposes to thrombosis. It has been suggested that the type of suture does not influence the success rate in the adult (Egdahl and Hume, 1956), although it is of importance in the growing animal (Sauvage and Wesolowski, 1955). Our experience confirms that, although the suture type is unimportant in vein grafts, it is critical in woven teflon, and hence probably other fabric prostheses, where a continuous mattress suture appears to give better results than an over-and-over suture.

The success rate in this series increased with experience, suggesting that knowledge of the techniques of handling veins is probably the single most important factor in the success or failure of grafts in the venous system.

Table II.—Analysis of Results of Replacement of the Canine Inferior Vena Cava with Renal Vein Anastamosis

A. Autogenous Vein Compilation Replacement

Patent		*Closed*	
Follow-up Period	Reasons for Death	Follow-up Period	Reason for Death
1. 36 hours	Pneumonia	1. 36 hours	Pneumonia
2. 36 hours	Pneumonia	2. 36 hours	Pneumonia
3. 2 days	Hæmorrhage	3. 2 days	No cause found
4. 3 days	Pneumonia	4. 6 days	No cause found
5. 7 days	Evisceration	5. 9 days	Sacrificed
6. 24 days	Sacrificed	6. 18 days	Sacrificed
7. 41 days	Sacrificed		
8. 44 days	Sacrificed		
9. 58 days	Sacrificed		
10. 107 days	Sacrificed		
11. 118 days	Sacrificed		
12. 120 days*	Sacrificed		
13. 193 days	Sacrificed		

* This graft, though patent with a normal renal vein, was fibrosed with constrictions at both upper and lower anastomoses.

B. Crimped Woven Teflon Replacement

Patent		*Closed*	
Follow-up Period	Reason for Death	Follow-up Period	Reason for Death
1. 13 days	Sacrificed	1. 3 days	Widespread thrombosis
2. 40 days	Sacrificed	2. 7 days	Sacrificed
3. 47 days	Sacrificed	3. 14 days	Sacrificed
4. 143 days	Sacrificed	4. 107 days	Sacrificed

Aortic homografts, and to a lesser extent fabric prostheses, are easy to place in the venous system, but they tend to wrinkle and furrow because the low intraluminal pressure in the veins is not great enough to maintain their shape. The work of Botham and others (1960) with porous sponge teflon and our own with crimped woven teflon suggests that acceptable results may be obtained with a flexible prosthesis that is not easily deformed. However, mural thrombosis, which appears inevitable in plastic prostheses, tends to propagate and occlude the graft because of the low rate of flow. Although autogenous vein grafts, like the veins themselves, are difficult to handle surgically, the difficulties can be overcome by much practice and careful attention to technique. They should be preferred since they have many advantages. They are usually maintained in the distended state by the existing low venous pressures. They are available when needed, because there are a number of suitable veins, such as the superficial femoral or saphenous veins, which can be excised without jeopardizing the patient's circulation. If the grafts remain open in the immediate post-operative period, there is a likelihood that they will remain patent indefinitely, because as autogenous materials they are immunologically inactive. The diameter of the vessel to be replaced can be accurately matched by compilating two or three veins to form a tube of any desired size within the limits of possible clinical need.

SUMMARY

1. The clinical indications for replacement of the great veins have been reviewed. They are concerned with the relief of superior vena cava syndrome and operations on the portal system, venous injury, and arteriovenous fistula.

2. Experimental studies on venous replacement have been summarized. Recent workers have recommended autogenous veins from the saphenous or superficial femoral system for this purpose.

3. In this study the inferior vena cava of the dog was replaced by compilated autogenous vein grafts and teflon prostheses. The renal vein has been anastomosed to the graft in order to use the viability of the kidney as an indication of graft patency.

4. The results have emphasized the advantages of autogenous vein grafts in the replacement of the venous system provided meticulous technique is employed.

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THE EFFECT OF THE ANTIHISTAMINES ON THE ANTRAL PHASE OF GASTRIC ACID SECRETION IN DOGS

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THE UNIVERSITY DEPARTMENT OF SURGERY, ROYAL INFIRMARY, SHEFFIELD

ALTHOUGH parenterally administered histamine is known to be a potent stimulus to acid secretion by the stomach, debate continues concerning its precise site of action and its possible role as the final common pathway for all forms of gastric secretion, whether stimulated physiologically or pharmacologically. The finding that the antihistamines have been shown to antagonize almost all histamine effects save that on acid gastric secretion has cast considerable doubt on a possible physiological role of histamine in acid secretion by the stomach.

Suspecting that the anomaly might be due to failure of the antihistamine to reach the acid-secreting cells in adequate concentration, Kay and Forrest (1956a,b; 1957; 1958) applied the antihistamine locally by instillation into separated gastric pouches in dogs and showed an apparent specific inhibitory effect of the drug on parietal-cell activity. This conclusion was at variance with that of Robertson and Grossman (1952), who suspected a non-specific effect of the antihistamines. Ragins, Benditt, Greenlee, and Dragstedt (1958) have since demonstrated that the antagonism of locally applied promethazine to histamine-stimulated gastric secretion is a toxic and not a pharmacological effect. This has subsequently been confirmed by Blair and Forrest (1960).

In the course of the studies reported by Ragins and others (1958), the new observation was made—namely, that the antral phase of acid secretion in dogs could be inhibited by the intravenous administration of promethazine or neo-antergan, two potent antihistamines. On the basis of this finding, these investigators suggested tentatively that histamine is involved in the mediation of the secretory response to antral stimulation.

In addition to antagonizing the action of histamine, the antihistamines are known to possess other pharmacological effects; these drugs are able, in varying degrees, to block cholinergic nerve impulses (atropine-like action). Atropine has been shown to reduce the gastric secretory response to the ingestion of a meal (Code, 1951). The present investigation was planned to test the hypothesis that the action

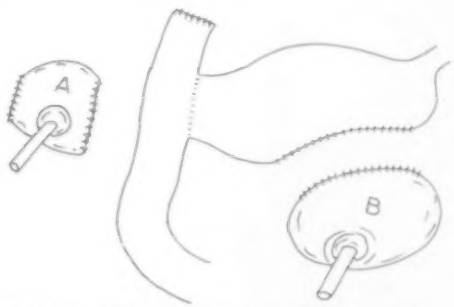

FIG. 796.—Diagram to show the isolated antral pouch (A) and separated (Heidenhain) pouch (B).

of certain antihistamines on the antral phase of acid secretion can be explained by their atropine-like activity.

In a first group of experiments we studied the effect of promethazine, mepyramine, and atropine on the antral phase of acid secretion in dogs. In a second group of experiments we investigated the effect of a new antihistamine (R.D. 1836) which has an antihistaminic potency equivalent to that of promethazine hydrochloride, but with insignificant atropine-like activity.

The evidence presented in this paper supports the view that the antihistamines do not have a specific effect on the antral phase of gastric acid secretion, any observed inhibition being explicable by an atropine-like action.

* In receipt of full-time Research Assistant Grant from the Medical Research Council.

METHOD OF STUDY

Three dogs were used throughout. Each was prepared with two pouches—a separated pouch of the body of the stomach (Heidenhain type) and an isolated antral pouch (*Fig.* 796). After operation, at least three weeks elapsed before tests were made.

The dogs were fasted for 16 hours before each experiment, which was conducted as follows: The antral pouch was stimulated continuously by perfusion with a 3 per cent suspension of desiccated liver in water, the *p*H of the suspension being adjusted to 7·0. This form of stimulation provoked the secretion of acid from the Heidenhain pouch and the output of acid soon reached a fairly constant level. The acid secreted from the Heidenhain pouch was collected at intervals of 15 min., the volume of each sample measured and the concentration of the acid determined by titration with 0·1 *N* sodium hydroxide, using Topfer's solution as indicator. From these determinations the output of free acid in mEq. was calculated. When collections over a control period of 90 min. had been made, the drug to be tested was given as a single intravenous injection.

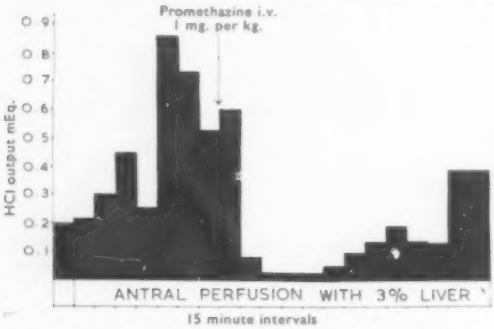

FIG. 797.—To illustrate the inhibitory effect of promethazine, 1 mg./kg., on the antral phase of gastric acid secretion.

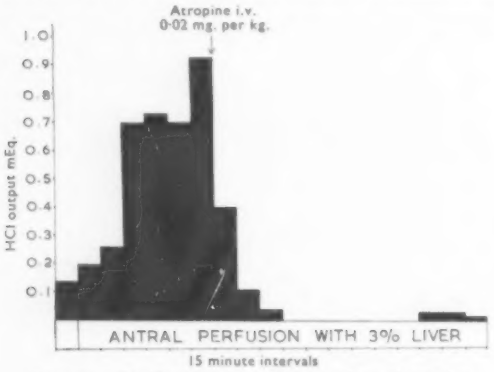

FIG. 799.—To illustrate the inhibitory effect of atropine, 0·02 mg./kg., on the antral phase of gastric acid secretion.

In a first group of experiments the dosage of the drugs employed was promethazine hydrochloride 1 mg./kg. body-weight, mepyramine maleate 5 mg./kg. body-weight, and atropine sulphate 0·02 mg./kg. body-weight. The dosage of the antihistaminic drugs was chosen so that each would have approximately the same antihistaminic potency; mepyramine is known to have relatively slight atropine-like activity (Loew, 1950). The dose of atropine (0·02 mg./kg.) was selected to give a cholinergic blocking effect approximately equal to that of 1 mg./kg. promethazine (Dobkin, Wyant, and Aasheim, 1958). Each drug was tested on three separate occasions on each of the three dogs.

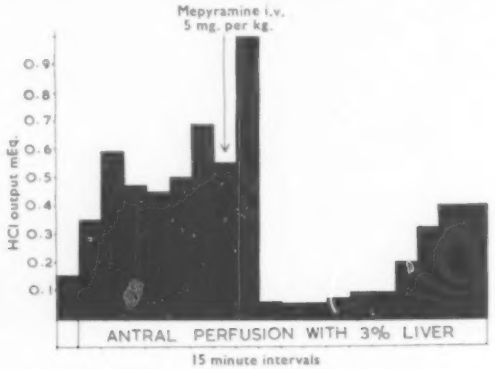

FIG. 798.—To illustrate the inhibitory effect of mepyramine, 5 mg./kg., on the antral phase of gastric acid secretion.

In a second group of experiments, when the above study was well advanced, the effect of a new antihistamine (R.D. 1836) was studied. This compound had been shown to have negligible anticholinergic activity and an antihistamine effect equivalent to that of promethazine hydrochloride, dose for dose. Consequently, R.D. 1836 was injected intravenously in a dose of 1 mg./kg. body-weight under precisely the same conditions of experiment as had been employed for the comparison of promethazine, mepyramine, and atropine. The same three dogs were used and four tests were made on each.

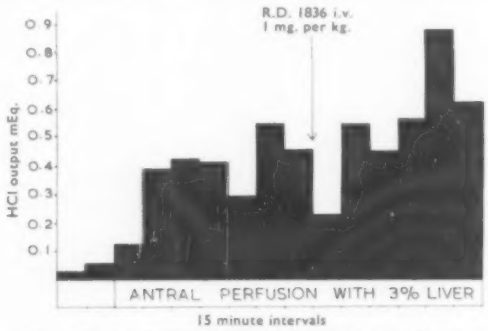

FIG. 800.—To illustrate the inhibitory effect of R.D.1836, 1 mg./kg., on the antral phase of gastric acid secretion.

RESULTS

The Effect of Promethazine, Mepyramine, and Atropine on the Antral Phase of Acid Gastric Secretion.—Promethazine, mepyramine,

and atropine each reduced the output of acid from the Heidenhain pouch in all of the tests (*Table I*).

The period of inhibition from a single injection of *promethazine* ranged from 90 min. to 3 hr. and was usually complete for about 45 min. A representative experiment (Dog A) is shown in *Fig.* 797.

The inhibition following a single injection of *mepyramine* in Dogs A and C tended to be less complete and of shorter duration; a representative experiment (Dog A) is illustrated in *Fig.* 798. Unfortunately, Dog B was extremely sensitive to the dosage of mepyramine employed in these studies and one experiment had to be abandoned owing to convulsions and the need to restrain the animal. It will be noted that, in contrast to the results obtained in Dogs A and C, a single injection of mepyramine produced considerable inhibition in Dog B, but, as all three experiments were associated with obvious side-effects, it seems likely that this inhibition can be explained by the toxic effects of the drug in this particular animal.

Table I.—The Effect of Promethazine, Mepyramine, and Atropine on the Acid Output from Heidenhain Pouches during Antral Stimulation

Mean Output of HCl in 3 Dogs during Irrigation of Antrum with Liver Suspension

(mEq. per 90-min. period)

Dog	Promethazine 1 mg. kg. i.v.		Mepyramine 5 mg. kg. i.v.		Atropine 0·02 mg. kg. i.v.	
	Before	*After*	*Before*	*After*	*Before*	*After*
A	3·365	0·414	3·240	1·067	2·750	0·276
B	1·334	0·388	1·619*	0·125*	1·570	0·261
C	0·923	0·159	1·367	1·132	0·910	0·201

Each figure represents the mean of 3 tests
* Mean of 2 tests only

Table II.—A Comparison of the Inhibitory Effect of Intravenous Promethazine, Mepyramine, and Atropine on the Acid Output from Heidenhain Pouches during Antral Irrigation with Liver Suspension in 3 Dogs

	No. of Tests	Mean Percentage Reduction in Acid Output (90-min. periods before and after drug)
Promethazine hydrochloride (1 mg. kg.)	9	81
Mepyramine maleate (5 mg. kg.)	8	56
Atropine sulphate (0·02 mg. kg.)	9	82

Atropine, like promethazine, caused complete inhibition of acid secretion in some specimens and the duration of the inhibitory effect tended to be greater than that following the antihistamine; a representative experiment (Dog A) is illustrated in *Fig.* 799.

A comparison of the inhibitory effect of the three drugs on the acid output from the Heidenhain pouches in response to antral irrigation with a liver suspension is given in *Table II*. In 9 tests with promethazine hydrochloride the mean percentage reduction in acid output was 81 per cent; this does not differ from the 82 per cent reduction obtained in the 9 experiments using atropine sulphate; these two drugs were given in a dosage selected to have equal atropine-like activity. By contrast, the mean reduction in acid output in the 8 experiments using mepyramine maleate was 56 per cent. This reduction is of a different order from those observed with the other two drugs.

The Effect of R.D. 1836 on the Antral Phase of Acid Gastric Secretion.—In all 12 tests R.D. 1836 failed to have any significant effect on the acid output from the Heidenhain pouches (*Table III*). A representative experiment in Dog A is given (*Fig.* 800).

Table III.—The Effect of Intravenous R.D. 1836 on the Acid Output from Heidenhain Pouches during Antral Stimulation

Mean Output of HCl in 3 Dogs during Irrigation of Antrum with Liver Suspension

(mEq. per 90-min. period)

Dog	Before R.D. 1836 (1 mg. kg.)	After R.D. 1836 (1 mg. kg.)
A	3·919	4·200
B	2·462	2·866
C	1·145	1·151

Each figure represents the mean of 4 tests

DISCUSSION

Our studies have confirmed the observations of Ragins and others (1958), namely, that certain antihistamines given by the intravenous route inhibit the gastric phase of acid secretion in dogs. However, our results also show that when promethazine and mepyramine are given in a dosage to ensure equal antihistaminic potency, the former has a more profound inhibitory effect on the acid output of the Heidenhain pouches. In this dosage the atropine-like activity of promethazine is greater than that of mepyramine. This finding suggests, but does not prove, that the cholinergic blocking action of the antihistamines is an important factor in causing the observed inhibition of acid secretion.

On the basis of antisialogogic tests (Dobkin and others, 1958), 0·02 mg. atropine and 1 mg. promethazine have equal cholinergic blocking potency. In this dosage each reduced the acid output from the Heidenhain pouches to a similar degree. This is further evidence that promethazine inhibits the antral phase of secretion by an atropine-like and not by a specific antihistaminic action.

Reference has been made in our results to the fact that atropine, while depressing acid secretion to a similar extent as promethazine, tended to have a slightly more prolonged effect. This may well be due to the fact that atropine provides a specific anticholinergic block whereas promethazine has various other actions in addition to its ability to block cholinergic impulses. For example, histamine antagonists are themselves capable of releasing histamine (Schachter, 1952) and so may actually stimulate acid secretion to some extent. If this is so, it could explain the discrepancy between the duration of action of the two drugs.

The results of the first group of experiments led us to believe that proof of the hypothesis to be tested could best be obtained by conducting a similar series of experiments using an antihistamine possessing negligible atropine-like properties and we were fortunate enough to have the opportunity to study a new antihistamine (R.D. 1836) which satisfied this requirement. This drug, which has an antihistaminic potency equivalent to that of promethazine dose for dose, failed to reduce the acid output from the Heidenhain pouches secreting in response to antral stimulation. In conjunction with the knowledge that R.D. 1836 has negligible cholinergic blocking activity, this finding provides good evidence that antihistamines capable of inhibiting the antral phase of acid secretion in dogs do so by virtue of their anticholinergic property and not by a specific antihistaminic action.

SUMMARY

Three dogs were prepared with two pouches of the stomach—an antral pouch and a denervated pouch of the gastric body. Irrigation of the antral pouch with a suspension of desiccated liver produced an acid secretory response from the Heidenhain pouch.

Promethazine hydrochloride, an antihistamine with considerable atropine-like properties, inhibited acid secretion from the Heidenhain pouch to a greater degree than did the more specific mepyramine maleate.

The inhibitory effect of promethazine was similar to that of an equivalent dose of atropine sulphate.

R.D. 1836, a potent antihistamine with negligible atropine-like properties, failed to inhibit the antral phase of acid secretion.

It is concluded that any inhibition of the antral phase of acid gastric secretion following the parenteral administration of antihistamines results from the anticholinergic property of the antihistamine used. There is no evidence to support a specific action of the antihistamines on acid gastric secretion of antral origin.

Acknowledgement.—We are indebted to Boots Pure Drug Co. Ltd. for supplies of R.D. 1836 and for the pharmacological data relating to this compound.

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SHORT NOTES OF RARE OR OBSCURE CASES

PERFORATED EMPYEMA OF THE GALL-BLADDER IN AN INFANT

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ACUTE gall-bladder disease from any cause is rare in infancy. The successfully treated patient reported below is believed to be the youngest yet described. Downie (1938), from this same hospital, described a 15-month-old infant with acute cholecystitis which was treated by simple drainage of the gall-bladder. There was no suggestion of common-duct obstruction in his case, and the child made a good recovery without further interference. In older children acute cholecystitis has been described as a complication of scarlet fever (Swing and Bullova, 1938) and typhoid (Reid and Montgomery, 1920).

CASE REPORT

The patient, a female infant, was admitted to hospital at the age of 8 weeks. She was the second child of healthy parents. At birth she weighed 6 lb. and appeared to be a normal full-term baby.

She thrived until 4 weeks old when she became somewhat constipated, developed slight abdominal distension, and showed a tendency to vomit some of her feeds. These symptoms persisted and she was first seen as an out-patient at 7 weeks old. She had a fever of 101° F. The abdomen was noted to be moderately distended. No other abnormality was found and she was observed for a few days, during which time she appeared to recover completely.

Two days before her admission she first became jaundiced and her stools became pale and urine dark. There was a return of abdominal distension and she again began to vomit. On examination she was obviously an ill baby, weight 8 lb. 12 oz., temperature 98° F. She was deeply jaundiced. The abdomen was distended and palpation revealed a large mass on the right side, firm and smooth in outline, extending from the costal margin down into the iliac fossa. On rectal examination the apex of the mass could be felt outside the rectum anteriorly on the right side. X-ray examination of the abdomen confirmed the presence of this right-sided large mass which was extending down to the pelvis and displacing the stomach and intestines to the left. No calcification could be seen in it. Several loops of intestine appeared to be distended (*Fig.* 801). Hæmoglobin was 62 per cent, 9·15 g. per cent; white blood-cells, total count 20,000 per c.mm., of which 13,600 were polymorphonuclear leucocytes, many of them

immature, thus indicating a marked 'shift to the left'. Serum-proteins: total 6·4 g. per cent, albumin 2·7 g. per cent, globulin 3·7 g. per cent. Serum-bilirubin: 3·3 mg.

FIG. 801.—Radiograph of the abdomen showing right-sided large mass displacing stomach and intestines.

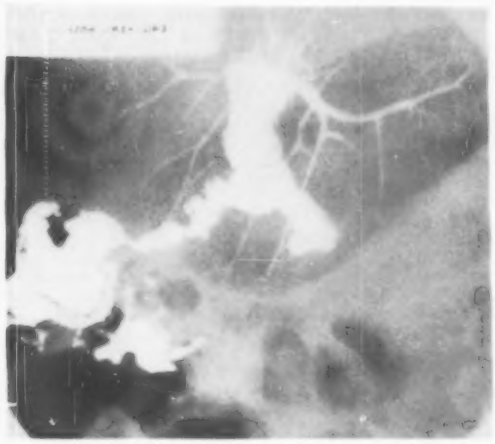

FIG. 802.—Cholangiogram, showing dye passing along cystic duct into the distended common bile-duct, which is completely obstructed near its lower end.

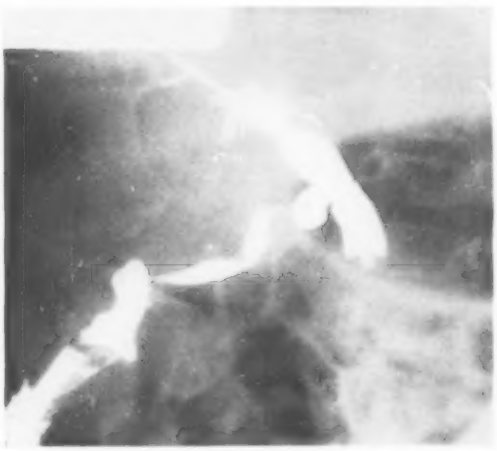

FIG. 803.—Cholangiogram two weeks later, showing obstruction to be unchanged.

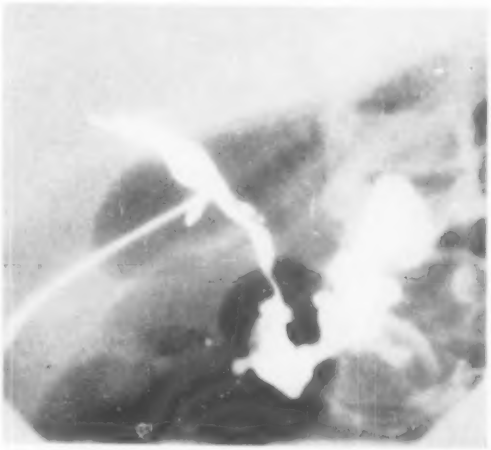

FIG. 804.—Cholangiogram performed after second operation. The common duct is no longer dilated, and the dye runs freely in to the duodenum.

per cent, serum-alkaline phosphatase: 34 King Armstrong units.

During the next twelve hours the baby had two large vomits and the abdominal distension greatly increased. The bowels were not open. A clinical diagnosis was made of obstructive jaundice combined with intestinal obstruction. The nature of the mass remained uncertain, but urgent laparotomy was considered necessary.

AT OPERATION.—A right paramedian incision was made. There was a large inflammatory mass closely adherent to the anterior parietal peritoneum. Free, thick, bile-stained pus was leaking into the peritoneal cavity at the inferior pole of the mass low in the right iliac fossa. The mass was carefully separated from the peritoneum and it was then realized that this was an enormous empyema of the gall-bladder, which had perforated at its fundus. A loop of ileum had become attached to the region of the perforation and the small intestine was almost completely obstructed at the ileocæcal junction by dense adhesions in this region. There was gross small-bowel distension. The adhesions were all separated. A self-retaining catheter was inserted into the gall-bladder through the fundal perforation and anchored by a double purse-string suture of catgut. The tube was brought out through a stab

incision. A swab of the pus was taken for culture. The abdomen was closed, a further drain being placed to the right iliac fossa.

PROGRESS.—Post-operative progress was stormy, but a slow improvement was maintained. Culture of the pus yielded a heavy growth of coliform bacilli and a coagulase-positive *Staph. aureus*. Full doses of the appropriate antibiotics were given. A wound infection healed slowly. The bile draining from the cholecystostomy gradually became clear. Bile-salts were given orally to compensate for this loss. Eleven days after operation a cholangiogram was obtained by instilling contrast medium down the catheter into the gall-bladder (*Fig.* 802). This showed that the cavity around the catheter head was no longer very large, and that from it the dye passed along the cystic duct and back into the hepatic duct system. It also passed down the common duct as far as a point of obstruction that was concave downwards. The nature of this common-duct obstruction remained obscure. Investigations revealed no evidence of a hæmolytic anæmia which might have resulted in stone formation. That the duct had at one time been patent was certain, as jaundice had only developed after eight weeks of life. Attempts to flush out with saline any obstructing biliary 'mud' were unsuccessful (Gross, 1953), and the baby continued to drain 90–150 ml. of bile daily whilst the stools remained clay-coloured. A further cholangiogram (*Fig.* 803) two weeks later showed the obstruction to be unchanged. As her general condition was now excellent it was decided to perform a further exploration. This was done six weeks after the initial laparotomy.

SECOND OPERATION.—The previous wound was re-opened. There were some adhesions around the gall-bladder and under-surface of the liver and the gall-bladder was still mildly inflamed. It was separated from the site of the external fistula. A narrow, malleable probe was passed down through the gall-bladder and cystic duct into the common duct until it passed behind the first part of the duodenum and seemed to have entered the second part. The second part of the duodenum was opened opposite the point of the probe, which was found to be still covered with mucous membrane. The probe was pushed through this, releasing a small quantity of thick, dark, inspissated material. The opening was further enlarged with a fine knife until it would take a large metal bougie. Fresh bile was now seen to be entering the duodenum quite freely. The duodenum was closed with a double layer of catgut sutures. A fine catheter was again sutured into the gall-bladder through the fundal opening. The abdomen was closed.

PROGRESS.—Post-operative progress was very rapid. A further cholangiogram was performed on the third day (*Fig.* 804). This showed that the common duct was no longer dilated and that there was free passage from the tube in the gall-bladder down into the duodenum. The tube was removed on the eighth day. The biliary fistula soon closed. The baby was discharged home fifteen days after operation.

She was followed up as an out-patient and was noted to be making excellent progress. Weight-gain was rapid, and general development appeared to be normal.

DISCUSSION

The case described above is that of an acute gall-bladder infection associated with obstruction of the common bile-duct. As jaundice did not develop until the infant was 8 weeks old this obstruction must initially have been incomplete. The existence of cases of incomplete congenital biliary obstruction, where subsequent complete blockage with inspissated mucus or bile occurred, was first stressed by Holmes (1916), who presented a detailed study of more than 100 cases of biliary abnormalities and showed that many of these were anatomically amenable to surgical treatment. Indeed, he quoted a case of Rotgans (1913) in which the anatomical abnormality must have been very similar to that in the present case. A 5-year-old girl had first developed attacks of colicky abdominal pain, with clay-coloured stools and pigmented urine after the age of 1 year. These attacks persisted for 4 years. At operation the gall-bladder and cystic and common ducts were dilated, and there appeared to be an obstruction at the ampulla of Vater. An external fistula was created and for a time all the bile drained through this. Four months later a side-to-side choledochoduodenostomy was performed and the girl made a full recovery. This is believed to be the earliest recorded case treated successfully.

Following Holmes's paper Ladd began to explore all cases of congenital biliary obstruction and in 1928 he described his first successful case. Since then exploration has become the generally accepted policy in these cases and many large series have been reported. Gross (1953) claims an overall success rate of 26 per cent of 198 babies treated medically and surgically for obstructive jaundice in the early months of life.

It is surprising that in the many recorded cases there are not more like the present one, in which acute infection occurred as an early complication of the biliary obstruction.

SUMMARY

A case of perforated empyema of the gall-bladder in an 8-week-old infant is reported. Emergency drainage of the gall-bladder was performed, and subsequent cholangiography showed an obstruction of the lower end of the common bile-duct. This was successfully relieved by transduodenal sphincterotomy.

The comment is made that in the very many reported series of congenital biliary obstruction there are few in which acute gall-bladder infection supervenes, and no record has been found of a similar case of this age.

My thanks are due to Dr. O. Wolff, under whose care this patient was admitted, for permission to publish this report; to Mr. F. Welsh for allowing me to perform the first operation and to assist with the second; and to Dr. R. Astley for the radiographs.

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PYELO-URETERITIS CYSTICA

By J. McARTHUR

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THE first recorded description of pyelo-ureteritis cystica was that of Morgagni (1761), who reported his post-mortem findings on a patient who had suffered from chronic urinary obstruction. In 1929 the condition was first diagnosed radiologically by Joelsen, but since then the number of reported cases has been small. McNulty (1957) reviewed 31 published cases, but of these the diagnosis was not adequately established in 15 patients, while more recently Veiga-Pires (1959), in reporting 2 cases, found only 32 other fully described cases in the literature. The case which is now reported illustrates the typical radiological and pathological findings and presented the common complication of the condition.

FIG. 805.—Retrograde pyelogram showing the radiolucent shadows in the left ureter.

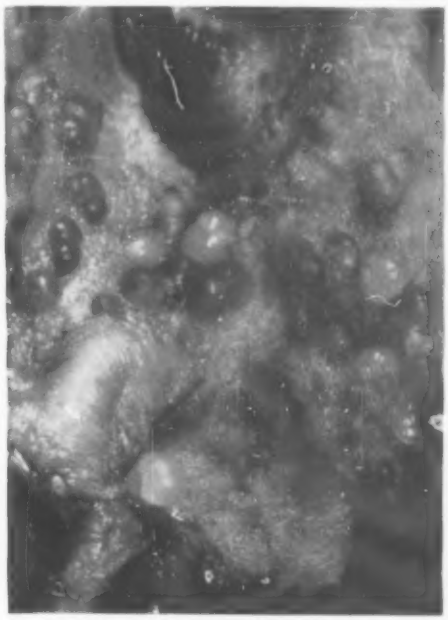

FIG. 806.—Photograph showing the cysts within the renal pelvis and at the pelvi-ureteric junction.

CASE REPORT

The patient was a married woman aged 60 years who complained of recurrent attacks of pain in the left loin of 4 years' duration. The attacks were fairly severe in nature and were associated with nausea, shivering, and sometimes vomiting. She had suffered from increased frequency of micturition during attacks but had no dysuria and no hæmaturia. Initially, the attacks occurred at intervals of 3–4 months and lasted for some 2 days, but more recently she had experienced trouble every 4–6 weeks and lasting for some 2 weeks.

ON EXAMINATION.—The patient was a rather pale woman of average build. There was no evidence of recent weight-loss. Abdominal examination showed her to be tender in the left loin and an indefinite cystic mass was palpable in this site. The blood-pressure was 190/90 mm. Hg. No other abnormality was noted on clinical examination. The hæmoglobin was 90 per cent; blood-urea 34 mg. per cent; and E.S.R. 15 mm. in the first hour (Wintrobe).

Intravenous pyelogram: This showed that in the right kidney function was normal and no abnormality was noted. No function was present in the left kidney.

Cystoscopy and retrograde pyelography: The bladder capacity was 400 ml. The base and trigone were moderately injected, but otherwise the bladder was normal. Both ureters were catheterized to 20 cm. without difficulty. Urine specimens were collected from the bladder and the right ureter. No urine was obtained from the left ureter. The bladder urine showed a heavy deposit of pus cells, and culture yielded a profuse growth of *Bact. coli*, highly sensitive to nitrofurantoin, chloramphenicol, streptomycin, and tetracycline, but only relatively poorly to sulphafurazole. There were no pus cells in the specimen from the right ureter and the culture from this side was sterile. In the retrograde film (*Fig.* 805) the right kidney and ureter were normal, but the left kidney appeared to be hydronephrotic and the left ureter was studded with numerous radiolucent shadows.

AT OPERATION (Dec. 8, 1959).—The left kidney was exposed through a lumbar incision. The kidney and renal pelvis were grossly dilated and the renal parenchyma reduced to a thin shell. There was no hydro-ureter and though the ureter felt a little irregularly thickened it was not macroscopically abnormal. The kidney and proximal two-thirds of the ureter were removed.

PATHOLOGY REPORT (*Fig.* 806).—The kidney is enlarged, being 16 cm. long and 15·5 cm. wide, excluding the pelvis, which is grossly dilated and is 11·5 cm. long and up to 6 cm. wide. A probe passes easily along the ureter into the renal pelvis. The pelvis contains thin blood-stained urine. The epithelial lining of the ureter bears numerous cystic projections 2–3 mm. in diameter. There are several of these cysts at the pelvi-ureteric junction. One

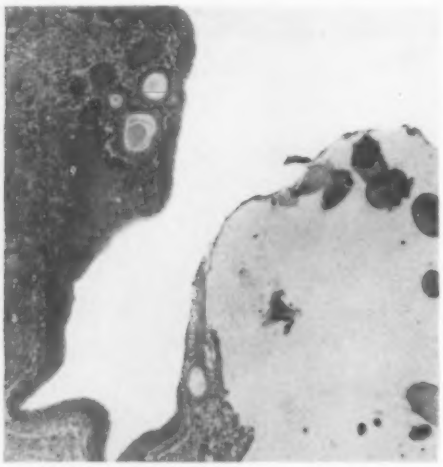

FIG. 807.—Photomicrograph showing the formed cyst and the changes in the ureteric wall.

is projecting over the mouth of the ureter; it is easily pushed aside by the probe but may well have constituted a bar to the outflow of urine. Jelly-like nodules up to 0·5 cm. in diameter stud the surface of the renal pelvis, being most numerous at its lower pole. The calices are greatly dilated and the renal parenchyma is reduced to a rim a few millimetres thick for the most part, but increases to 0·5 cm. at the centre of the kidney.

Photomicrograph (*Fig.* 807).—The wall of the renal pelvis and the ureter shows fibrous thickening. There is a dense infiltration by plasma cells beneath the epithelium and the epithelium forms small downward dipping processes many of which have become cystic. Some of these cysts are greatly distended and project above the surface. The kidney itself shows severe chronic pyelonephritis.

Diagnosis: (1) Ureteritis and pyelitis cystica. (2) Chronic pyelonephritis.

Convalescence was uneventful and the patient was dismissed home on Dec. 19.

DISCUSSION

Pyelo-ureteritis cystica is a disease affecting the older age-groups and occurring rather more frequently in females (62 per cent) than in males. The condition may be bilateral, but is more often confined to one side. It is generally agreed that pyelo-ureteritis cystica is usually associated with, and, indeed, consequent upon, a chronic urinary infection, and cystoscopy frequently shows evidence of a chronic cystitis which may be of the cystic type. The symptoms which arise from the disease may be minimal, but are usually those of the related urinary infection, though some authors (Stirling and Ash, 1941) consider hæmaturia to be a common feature of cystic disease. The diagnosis is made on the radiographic appearance of punched-out defects within the lumen of the contrast-filled ureter or renal pelvis, most numerous in the upper parts. Since the cysts act as obstructive agents within the ureter the condition is frequently complicated by the development of a hydronephrosis and treatment is dependent on the extent of this complication. When the kidney function is good and the degree of hydronephrosis moderate, the first aim must be to control the urinary infection. Thereafter an attempt can be made to destroy the cysts by the retrograde injection of a 2 per cent silver nitrate solution (Kindall, 1933; Kopp, 1946). However, when the hydronephrosis is gross and the condition unilateral, nephrectomy with ureterectomy must be undertaken for the relief of symptoms.

SUMMARY

A case of pyelo-ureteritis cystica is presented and the diagnosis and treatment of the condition are discussed.

I wish to thank Mr. R. L. Beveridge for permission to report this case and for his criticism of the paper. I would also thank Dr. P. M. Robb for the pathological report and Mr. A. A. Jenkins for the photography.

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REVIEWS AND NOTICES OF BOOKS

Medicine. A Lifelong Study. Edited by HUGH CLEGG, M.D., F.R.C.P. Editor of the *British Medical Journal*. Pp. 816 + xx. 1961. New York: World Medical Association. London: *British Medical Journal*. 105s.

THIS large volume contains the *Proceedings* of the Second World Conference on Medical Education held in Chicago in 1959. It is the second volume devoted to medical education, and it cannot fail to interest medical teachers the world over, for it provides medical opinion on this subject from some 66 countries and 179 medical schools.

Medical education has many facets, especially post-graduate medical education, and all these many facets have been considered in the various papers presented before this conference.

Sir George Pickering talks ably about the advanced training for general and specialty practice and the problem as to what is needed to encourage a man to continue to educate himself during the whole of his professional life; will he need educational boosts from time to time, and, if so, in what form and for how long? Many speakers have pointed out the serious consequences of divorcing the general practitioner from hospitals. This separation, more complete and permanent each day, has made it difficult, if not impossible, for the general practitioner to keep informed on medical advances, which have been so rapid in the last decades.

Sir Francis Fraser gives a very mature and practical lecture on the administrative and organizational methods and techniques useful in continuing medical education.

Victor Coppleson, of Australia, gives an excellent summary of the advanced training for general and specialty practice.

As a reference volume this book will be used in all countries where medical education exists.

The Management of Fractures and Soft Tissue Injuries. By the Committee on Trauma, American College of Surgeons. 8¼ × 5 in. Pp. 372 + xix. 1960. Philadelphia and London: W. B. Saunders Co. Ltd. 35s.

THE American College of Surgeons has brought together two valuable publications, one on the treatment of fractures, which has now been running for a number of years, and the more recent manual on soft-tissue injuries. The result is an extremely business-like book of reasonable length written by men of great experience and wisdom. The general style is, as formerly, simple and dogmatic, and at the present time, when accident surgery is becoming increasingly important, this is without question the ideal guide for the surgeon called upon to deal with injuries. Experts will disagree with some of the details, and in places, such as the section on major injuries of the thoracic cage, this manual is inadequate; but all will agree that if surgeons in casualty or accident departments took this book as their guide they would rarely go far wrong. It is a minor masterpiece.

Le cisti e i tumori primitivi del mesentere. By FRANCESCO FONTANA. 9½ × 6½ in. Pp. 130. Illustrated. 1959. Bologna: Cappelli Editore. No price given.

THE main interest of this dutiful labour carried out by an assistant at the Ospedale Maggiore, Bologna, is largely anecdotal, nearly half the book being devoted to detailed, illustrated records of twenty-one patients with solid tumours or cysts of the mesentery. All were operated upon; twenty by the Director (separately mentioned by name on twenty occasions) and one by an anonymous surgeon. The rest of the text consists of ruminations upon the corpus of approximately three hundred articles listed in the bibliography.

Parkinsonism, its Medical and Surgical Therapy. By IRVING S. COOPER, M.D., Ph.D., F.A.C.S. 11 × 8¾ in. Pp. 239 + xii, with 111 illustrations. 1961. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) £6 12s.

THE publication of this monograph on Parkinsonism should give new hope in the treatment of this distressing disease. During the last decade much progress has been made in the surgical treatment on both sides of the Atlantic. Since Putman in 1938 devised the operation of pyramidotomy in the cervical part of the spinal cord, many other operations have been devised at the level of the basal ganglia. Surgical occlusion of the anterior choroidal artery has also been performed by means of a metal clip. It is interesting to note that tremor, rigidity, and other components of the syndrome of Parkinsonism are often alleviated or abolished by this operation. Curiously, the best results have been achieved in far advanced and disabled cases.

The operative details of chemopallidectomy and chemothalamectomy are given in detail, and the author presents his findings in 1500 cases of basal ganglia surgery, surely a clinical record for any surgeon.

This book will take its place as the reference volume on the surgical treatment of Parkinsonism.

The Surgical Treatment of Portal Hypertension, Bleeding Esophageal Varices and Ascites. By M. JUDSON MACKBY, M.D., D.A.B.S., F.I.C.S. 10 × 6½ in. Pp. 250 + xviii. Illustrated. 1961. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 84s.

THE time is ripe for the publication of such a book as this, for the mass of surgical literature which has accumulated on this subject during the last decade is enormous.

Dr. Mackby has crystallized his thoughts and experience on the treatment of the surgical complications of cirrhosis. He has written a very practical and interesting monograph on this subject. His pictures illustrating the various techniques of surgical intervention are excellent and very clear.

The pathogenesis of portal hypertension, bleeding œsophageal varices, and ascites is well summarized, together with pathological physiology and some important biochemical features.

A useful extensive bibliography of the world literature on this subject is given at the end of the book. A most helpful reference volume.

Procedures in Vascular Surgery. By RICHARD WARREN, M.D., Clinical Professor of Surgery, Harvard Medical School. 10½ × 8 in. Pp. 211 + x, fully illustrated. 1960. London: J. & A. Churchill Ltd. 85s.

VASCULAR surgery came into its own during the second world war when surgeons had plenty of experience in dealing with vascular injuries of all magnitudes. With the introduction of arterial grafts another great stride in this branch of surgery took place.

Dr. Warren of Boston comes from a surgical centre which has always been to the fore in this surgical speciality. This book is full of sound surgical advice, it is lavishly illustrated, and well produced.

Various forms of treatment, from the reconstruction of coarctation to the stripping of varicose veins, are given,

and the author discusses the objectives, indications, and contra-indications, the pre- and post-operative preparations, and the actual operative procedure. The different forms of arterial grafting are well presented and beautifully illustrated. There is an excellent chapter dealing with the various amputations of the foot and ankle which may be required in obliterative arterial disease.

This book should prove of real value to all young surgeons who are preparing for higher surgical degrees or diplomas.

Praktische Operationskunde und Instrumentenlehre. By HARRO SEYFARTH and ELLY JAEGER. 9½ × 6½ in. Pp. 464 + xx, with 214 illustrations. 1960. Jena: Gustav Fischer. DM.36.70.

THOUGH texts devoted to operative surgery are plentiful, comparable works for the instruction of theatre staff are almost unknown. This book, a collaborative effort by an orthopædic surgeon and a theatre sister from two East German University Departments, seeks to make good just such a deficiency.

It deals with operative principles, theatre discipline, deployment of staff, and goes very fully into the sort of equipment, from furniture through instruments and suturing materials to dressings and drugs, to be found in a general operating theatre. In order to co-ordinate and channel all this vast fund of information, and to give some practical guidance, numerous standard operations are summarized, and the appropriate sets of instruments listed. In certain instances, as in the case of appendicectomy, graphic charts relate operative steps to requirement of instruments by the surgeon and his assistant.

Highly instructive, but not easily assimilable, even for the medically qualified, this book fulfils a very real need. It should make a good solid foundation on which to train the most fundamental, and often the most taken-for-granted member of the surgical team—the theatre sister.

Anatomy and Physiology for Radiographers. By J. E. BLEWETT, M.D., D.M.R., F.F.R., Director of Diagnostic Radiology Department, King's College Hospital, London, and the Belgrave Hospital for Children; and A. M. RACKNOW, B.Sc., M.B., D.M.R.E., Assistant Director of Diagnostic Radiology Department, King's College Hospital, London, and the Belgrave Hospital for Children. 8½ × 5½ in. Pp. 322. 22 Plates. London: Butterworth & Co. (Publishers) Ltd. 37s. 6d.

THIS is a book which will prove of real value to the many radiographers up and down the land, for it contains just the information, in two difficult subjects, which the modern radiographer requires. Both authors are matured teachers, and they have tackled the subject in a most practical way. It is refreshing to see such a wealth of good line drawings, which are lucid yet very simple; they certainly enhance the value of the book and make it quite outstanding.

There can be little doubt that this book will take its place as the standard work for the modern radiographer. The price is quite moderate.

Surgical Diseases of the Pancreas. By JOHN M. HOWARD, M.D., F.A.C.S., and GEORGE L. JORDAN, jun., M.D., M.S., F.A.C.S. 10 × 6½ in. Pp. 607 + xviii, with 199 illustrations. 1961. London: Pitman Medical Publishing Co., Ltd. £7 7s

THIS large and comprehensive work devoted to the surgical diseases of the pancreas deals with every aspect of the pancreatic problem, including medical and surgical management. Although Dr. Howard and Dr. Jordan have written the greater part of the book, they have called upon some of their surgical colleagues in various parts of America to contribute special chapters.

The problem of malignant disease of the pancreas is dealt with in a masterly fashion. Allen O. Whipple writes a stimulating article on "A Histological Sketch of the Pancreas".

Throughout the book the numerous illustrations are excellent and are beautifully produced. A full survey of the literature supports the text.

As a reference volume this book could not be bettered.

Atlas of Anatomy and Surgical Approaches in Orthopædic Surgery. By RODOLFO COSENTINO, M.D. Volume 2, The Lower Extremity. 11 × 8½ in. Pp. 264, with 139 illustrations. 1961. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) £5 12s.

THIS is the second volume of Dr. Cosentino's *Atlas*, and it is devoted to the surgical approaches to the lower extremity. It follows the same pattern as the first volume, which deals with the upper extremity. The 139 large plates revealing the anatomy of various orthopædic operations on the leg are of a high order, and cannot help but be of real value to the senior student and young surgeon who wish to have a reference volume to consult on such operations.

Professor Carroll B. Larson contributes a preface in which he endorses the value of the book.

Progress in Clinical Surgery. Edited by RODNEY SMITH, M.S., F.R.C.S. 9½ × 6 in. Pp. 327 + x, with 110 illustrations. 1961. London: J. & A. Churchill Ltd. 50s.

THE first edition of this book appeared in 1954, and much has happened in the field of surgery since then. The twenty-three contributors to this volume have done their work well, and the reader is given an excellent progress report in the many fields of surgery. The fields of cardiac and cerebral surgery have advanced enormously, and A. L. d'Abreu and Wylie McKissock have covered these fields in an admirable manner.

Other conditions such as hyperparathyroidism, thyrotoxicosis, Cushing's Syndrome, hiatus hernia, hepatectomy, hepatic coma, ectopia vesicæ, and transplantation of the ureters are considered in detail, and are very well presented.

The book will prove of real value to all who intend presenting themselves for the Final Examination for F.R.C.S., and the younger surgeons will profit by reading it.

Rodney Smith has picked his team well, and the result is a credit to British Surgery.

Visual Field Defects after Penetrating Missile Wounds of the Brain. By HANS LUKAS TEUBERS, WILLIAM S. BATTERSBY, and MORRIS B. BENDER. 10 × 6½ in. Pp. 143 + xii, with 55 illustrations. 1961. Cambridge, U.S.A.: Harvard University Press. (London: Oxford University Press.) 38s.

THIS monograph is devoted to an important subject, namely the visual field defects following war wounds of the brain. The authors have studied 203 patients with penetrating wounds of the brain, and of these 46 were found to have visual field defects on repeated tests extending over ten years after their wounding. The authors found that destruction in the presumed region of both occipital poles can lead to selective loss of the entire macular region; conversely, penetrating wounds through the posterior lobe substance directed somewhat more anteriorly may be followed by permanent bilateral hemianopia with macular sparing.

This investigation was carried out at the Psychophysiological Laboratory, New York University, Bellevue Medical Center, a unit devoted primarily to the study of after-effects of brain injury in man.

Neurologists and neurosurgeons will find this monograph of great practical value; all medical libraries should have a copy for reference.

Surgical Urology. By R. H. FLOCKS, M.D., and DAVID CULP, M.D. Second edition. 8¼ × 5½ in. Pp. 441, with 126 plates. 1961. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 79s.

THE first edition of this useful handbook of operative surgery dealing with the urinary tract was published some six years ago. The new second edition shows considerable revision, and several new illustrations have been added. A new section has been added which deals with isolated bowel segments in urological surgery.

The senior student and registrars will find this book very useful.

Cone of Oblivion. By L. J. LUDOVICI. 8½ × 5½ in. Pp. 224. 1961. London: Max Parrish & Co. Ltd. 25s.

THIS is an interesting account of the controversy which existed in America over who discovered ether anæsthesia. This book discusses the points in favour of William Morton, who demonstrated the use of ether anæsthesia in Massachusetts General Hospital in 1846, after having tried out its effect on dogs.

Charles Thomas Jackson—who laid claim to the discovery of ether anæsthesia—was obviously mentally deranged, and his behaviour just prior to his death proves this.

The author weighs up the pros and cons of this controversy very well.

Ring the Night Bell. By PAUL B. MAGNUSON. 8½ × 5½ in. Pp. 376. 1961. London: William Heinemann. 25s.

A SURGEON'S life is full of interest and adventure, and this is the reason why so many medical men write their own autobiography.

Paul B. Magnuson is no exception; he had an exciting and intriguing life, and he saw every facet of a surgeon's experience from first aid to major surgical operations. He met many of the great men of America during his years of work, and he was outstanding in his criticism when he thought this was justified.

An entertaining book, well written, and full of interest to both medical and lay public.

The Transplantation of Tissues and Organs. By MICHAEL F. A. WOODRUFF, M.D., M.S. (Melb.), F.R.C.S., F.R.C.S.E., F.R.A.C.S. 9¾ × 6½ in. Pp. 777 + xxxiv, with 196 illustrations. 1961. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) £10 4s.

THIS is a comprehensive work covering in a very real way the whole aspect of tissue and organ transplantation. The time is ripe for such a presentation as this, for much work has been done in the last decade both in this country and abroad.

The grafting of tissue has been in vogue for over fifty years, and since that time plastic surgery has been born, and the various plastic clinics have done much to improve transplantation of bone, cartilage, and other connective tissues.

The author of this work is a general surgeon who has devoted many years to research, in which he has been helped by biologists and other scientific investigators.

The first nine chapters of the book deal with basic biological problems relating to autotransplants, homotransplants, and heterotransplants of both normal and neoplastic tissue. The various methods of storing tissue are discussed.

Throughout the book the numerous illustrations are of a high order, and have been well produced.

The various operative techniques are well described and illustrated.

The last two chapters are devoted to pedicle transplantation and anastomosis of hollow viscera in reconstruction of the gastro-intestinal, biliary, pancreatic, and urinary tracts.

A most comprehensive bibliography is given at the end of the book as well as an extensive author index.

This work will take its place in world surgical literature, and become the standard reference volume on transplantation of tissues and organs.

A Synopsis of Physiology (Rendle Short). Edited by C. C. N. VASS, M.Sc., Ph.D., M.B., Ch.B., Reader in Physiology in the University of London. Fifth edition. 7¼ × 4¾ in. Pp. 360. Illustrated. 1961. Bristol: John Wright & Sons Ltd. 30s.

THE new fifth edition of *A Synopsis of Physiology* has been carefully revised and brought up to date. Much has happened in the field of physiology in the last twelve years, and many new facts have been established.

The chapter on the heart is outstanding, and the practical way in which the cardiac output is measured is to be commended, as well as the external work of the heart. It is great progress in the knowledge of the physiology of the heart which has made cardiac surgery possible. In fact, the cardiac surgeon of to-day must have a thorough knowledge of modern physiology.

Another outstanding and interesting chapter is that devoted to diet and growth; it contains the important formulæ of some of the vitamins.

Doctor Vass has done his work well, and he has produced a very valuable book for the student; to be able to condense the whole of physiology into some 358 pages is a difficult task.

The book is a credit to the well-known Synopsis Series, and as a quick revision book it could not be bettered.

The Closed Treatment of Common Fractures. By JOHN CHARNLEY, B.Sc., M.B., F.R.C.S. Third edition. 9¾ × 6½ in. Pp. 272 + xii, with 215 illustrations. 1961. Edinburgh and London: E. & S. Livingstone Ltd. 50s.

THE importance and popularity of this book are assured, for the first edition only appeared in 1950.

In this traumatic age fractures are all too common, and it is essential that students and practitioners alike should know how the common fractures are treated. Mr. Charnley's book deals with the conservative treatment of fractures, and he gives an excellent account of the various methods of treatment by splints and plaster-of-Paris, and skeletal traction. There are numerous illustrations throughout the book, and these have been well chosen, especially the radiographs, which are excellent.

This is a really worthwhile book for the resident medical officer and the registrar who has to treat fractures.

Angiography. Edited by HERBERT L. ABRAMS, M.D. In two volumes. 10¼ × 8½ in. Pp. 807, with 1345 illustrations. 1961. London: J. & A. Churchill Ltd. £17.

THIS large and comprehensive work in two volumes covers the whole extensive aspect of angiography. The volumes are extensively illustrated, in fact they might be termed an atlas of angiography. The volumes are of considerable size, and this allows for the reproduction of large radiographs, which is a distinct advantage.

There are some thirty-two contributors, all of whom have an international reputation in the field of radiology.

Probably no branch of radiology has advanced so extensively as that devoted to angiography. With the advent of new and improved opaque media, and of modern equipment for high-speed filming, angiography may be said to have come into its own.

The blood-supply to every organ of the body can now be visualized, and this work should prove of value—not only to clinicians, but also to students who are learning anatomy.

Dr. Abrams should be congratulated on an excellent production.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Fundamentals of Psychology: The Psychology of Thinking. Edited by ERNEST HARMS. Volume 91, Pp. 1–158 of *Annals of the New York Academy of Sciences.* 9 × 6 in. Dec., 1960. No price given.

Culture, Society, and Health. Edited by VERA RUBIN. Volume 84, Pp. 783–1060 of *Annals of the New York Academy of Sciences.* 9 × 6 in. Dec., 1960. No price.

Thermodynamics and Mechanics of Polymer Systems. Edited by F. R. EIRICH. Volume 89, Pp. 573–714 of *Annals of the New York Academy of Sciences.* 9 × 6 in. Jan., 1961. No price.

Gynäkologische Urologie. By W. LANGREDER. 10¼ × 6¾ in. Pp. 306 + xii, with 135 illustrations. 1961. Stuttgart: Georg Thieme Verlag. DM.59.

Les Ictères Post-Opérative Précoces. By F. POILLEUX and A. KAPANDJI. 9 × 7 in. Pp. 108, with 36 illustrations. 1961. Paris: L'Expansion Editeur. 14 NF.

La Radiculographie Lombo-Sacrée par Substance Iodée Hydrosoluble et Résorbable. By JACQUES FERRAND, JEAN ROBERT D'ESLOUGUES, and JACQUES BASSOTTI. 10 × 8¼ in. Pp. 108, with 82 illustrations. 1961. Paris: L'Expansion Editeur. No price given.

Tumors of the Female Sex Organs. By ARTHUR T. HERTIG, M.D., and HAZEL GORE, M.B., B.S. 10¼ × 7¾ in. Pp. 176, with 157 illustrations. 1961. Washington, D.C.: Armed Forces Institute of Pathology. $1.40.

Surgery is Destined to the Practice of Medicine. By Sir REGINALD WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S., F.R.A.C.S. (Hon.), F.R.C.S.E. (Hon.), F.A.C.S. (Hon.), Orthopædic Surgeon-Extra to Her Majesty the Queen, Orthopædic Surgeon to His Late Majesty King George VI. 10 × 6¾ in. Pp. 81, with 77 illustrations. 1961. Edinburgh: E. & S. Livingstone Ltd. 21s.

Histoire de la Chirurgie. By CLAUDE D'ALLAINES. 7 × 4 in. Pp. 128. 1961. Paris: Presses Universitaires de France. No price given.

Management of Fractures, Dislocations, and Sprains. By H. EARLE CONWELL, M.D., F.A.C.S., and FRED C. REYNOLDS, M.D. Seventh edition. 9¾ × 6 in. Pp. 1153, with 1227 illustrations. 1961. St. Louis: C. V. Mosby Co. (London: Henry Kimpton). £10 2s. 6d.

Surgical Diseases of the Chest. Edited by BRIAN BLADES, M.D. 9¾ × 6¾ in. Pp. 580, with 267 illustrations. 1961. St. Louis: C. V. Mosby Co. (London: Henry Kimpton). £8 5s.

Carcinoma of the Colon. By E. G. MUIR, F.R.C.S. 9 × 5½ in. Pp. 181, with 61 illustrations. 1961. London: Edward Arnold (Publishers) Ltd. 42s.

Die Chirurgische Behandlung der Angeborenen Fehlbildungen. By Dr. K. KREMER. 10¼ × 7¼ in. Pp. 592 + xvi, with 349 illustrations. 1961. Stuttgart: Georg Thieme Verlag. DM.118.

Injuries and Infections of the Hand. By ROBERT H. C. ROBINS, M.A., M.B., B.Chir., F.R.C.S. 9¾ × 7¼ in. Pp. 220 + xii, with 157 illustrations. 1961. London: Edward Arnold (Publishers) Ltd. 63s.

A Doctor's Memoirs. By A. J. WILLINSKY. 8¾ × 5½ in. Pp. 183. 1961. London: Macmillan & Co. Ltd. 28s.

Cardiopericardiomyopexy. By Dr. AARON N. GORELIK. 8½ × 5¼ in. Pp. 176, with 30 illustrations. 1961. New York: The Myopexy Association of New York. No price given.

Assessment of the Activity of Disease. By J. S. LAWRENCE, M.D., M.R.C.P., Director, the Empire Rheumatism Council Field Unit Based on the University of Manchester. 10 × 6¼ in. Pp. 252, with 34 tables and 37 illustrations. 1961. London: H. K. Lewis & Co. Ltd. 42s.

Neuroradiology Workshop. By LEO M. DAVIDOFF, M.D., HAROLD G. JACOBSON, M.D., and HARRY M. ZIMMERMANN, M.D. 11 × 8 in. Pp. 256, with 89 illustrations. 1961. New York and London: Grune & Stratton Ltd. $16.50.

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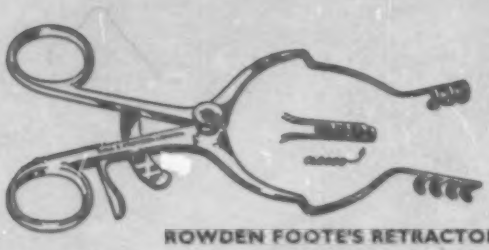

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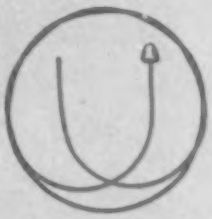

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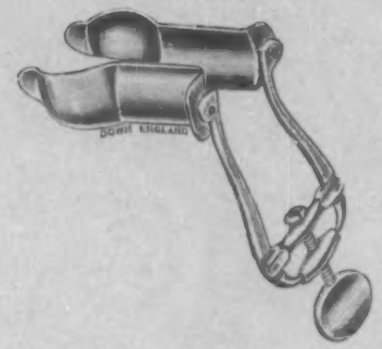

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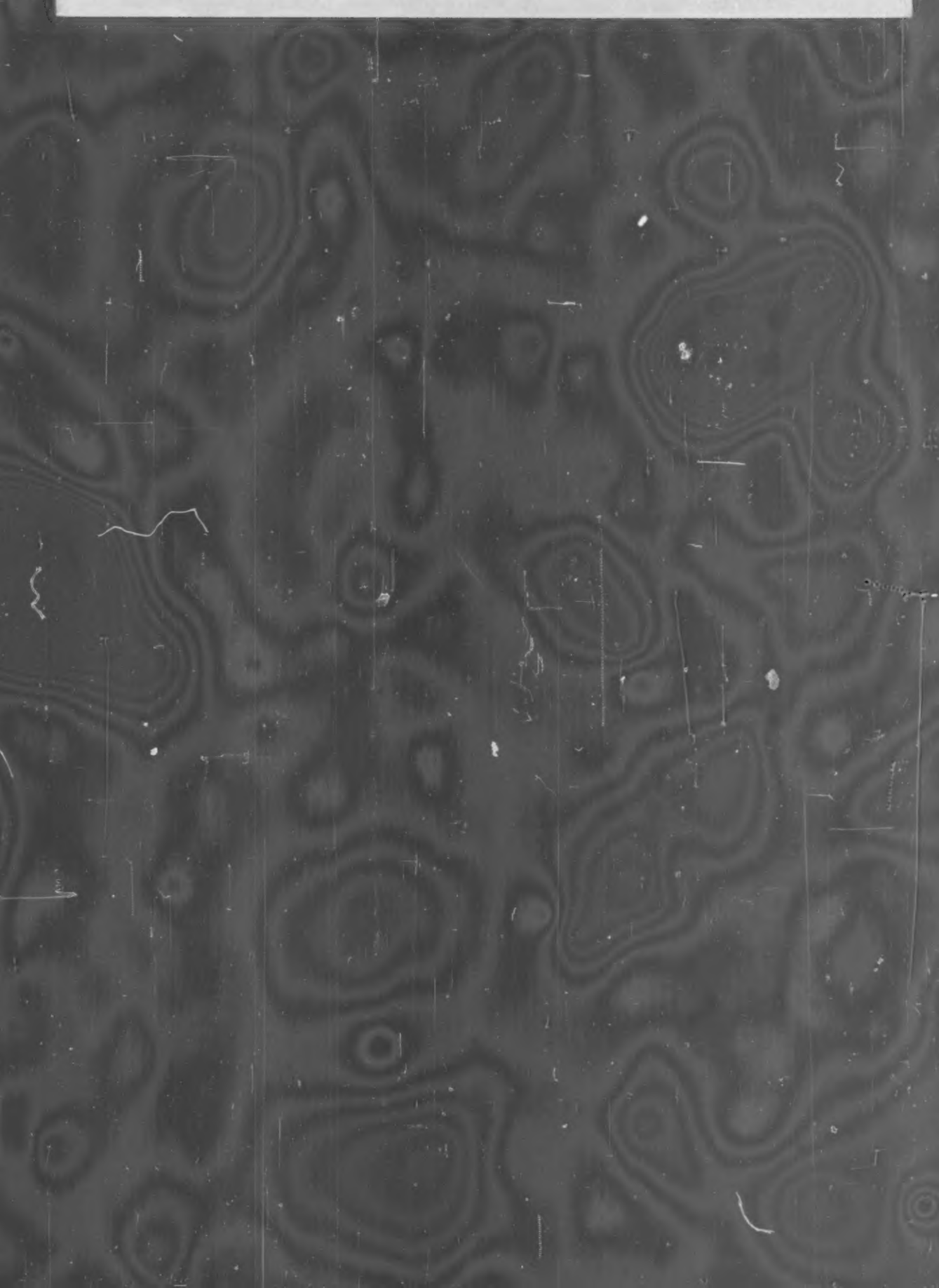
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